

COLOR ATLAS OF COMMON ORAL DISEASES

Fourth Edition



ROBERT P. LANGLAIS
CRAIG S. MILLER
JILL S. NIELD-GEHRIG

VISIT...

LANZAROTE
Caliente.COM

Color Atlas of Common Oral Diseases
Fourth Edition

Color Atlas of Common Oral Diseases

Fourth Edition

Robert P. Langlais, DDS, MS, FACD, FICD

Professor

Department of Dental Diagnostic Science
University of Texas Health Science Center at San Antonio
School of Dentistry
San Antonio, Texas

Craig S. Miller, DMD, MS, FACD

Professor of Oral Medicine

Department of Oral Health Practice and Department of Microbiology, Immunology and
Molecular Genetics
College of Dentistry, College of Medicine
University of Kentucky
Lexington, Kentucky

Jill S. Nield-Gehrig, RDH, MA

Dean Emeritus, Division of Allied Health
Asheville-Buncombe Technical Community College
Asheville, North Carolina



Wolters Kluwer | Lippincott Williams & Wilkins

Philadelphia • Baltimore • New York • London
Buenos Aires • Hong Kong • Sydney • Tokyo

Acquisitions Editor: John Goucher
Managing Editor: Andrea Klingler
Project Manager: Rosanne Hallowell
Manufacturing Manager: Margie Orzech
Marketing Manager: Allison Noplock
Creative Director: Doug Smock
Cover Designer: David Levy
Production Services: Aptara, Inc.

Fourth Edition
© 2009 by Lippincott Williams & Wilkins, a Wolters Kluwer business
530 Walnut Street
Philadelphia, PA 19106
LWW.com

Third Edition © 2003 by Lippincott Williams & Wilkins.

All rights reserved. This book is protected by copyright. No part of this book may be reproduced in any form or by any means, including photocopying, or utilizing by any information storage and retrieval system without written permission from the copyright owner, except for brief quotations embodied in critical articles and reviews.

Printed in China

Library of Congress Cataloging-in-Publication Data

Langlais, Robert P.
Color atlas of common oral diseases / Robert P. Langlais, Craig S. Miller,
Jill S. Nield-Gehrig. —4th ed.
p. ; cm.
Includes index.
ISBN 978-0-7817-8097-1
1. Mouth—Diseases—Atlases. 2. Oral medicine—Atlases. I. Miller, Craig
S. II. Nield-Gehrig, Jill S. (Jill Shiffer) III. Title.
[DNLM: 1. Mouth Diseases—pathology—Atlases. 2. Tooth Diseases—
pathology—Atlases. WU 17 L282c 2009]
RC815.L35 2009
617.5'2200222—dc22

2008039202

Care has been taken to confirm the accuracy of the information presented and to describe generally accepted practices. However, the authors, editors, and publisher are not responsible for errors or omissions or for any consequences from application of the information in this book and make no warranty, expressed or implied, with respect to the currency, completeness, or accuracy of the contents of the publication. Application of this information in a particular situation remains the professional responsibility of the practitioner.

The authors, editors, and publisher have exerted every effort to ensure that drug selection and dosage set forth in this text are in accordance with current recommendations and practice at the time of publication. However, in view of ongoing research, changes in government regulations, and the constant flow of information relating to drug therapy and drug reactions, the reader is urged to check the package insert for each drug for any change in indications and dosage and for added warnings and precautions. This is particularly important when the recommended agent is a new or infrequently employed drug.

Some drugs and medical devices presented in this publication have Food and Drug Administration (FDA) clearance for limited use in restricted research settings. It is the responsibility of health care providers to ascertain the FDA status of each drug or device planned for use in their clinical practice.

The publishers have made every effort to trace copyright holders for borrowed material. If they have inadvertently overlooked any, they will be pleased to make the necessary arrangements at the first opportunity.

To purchase additional copies of this book, call our customer service department at (800) 638-3030 or fax orders to (301) 223-2320. International customers should call (301) 223-2300.

Visit Lippincott Williams & Wilkins on the Internet at LWW.com. Lippincott Williams & Wilkins customer service representatives are available from 8:30 am to 6 pm, EST.

10 9 8 7 6 5 4 3 2 1

*To our spouses,
Denyse, Sherry, and Dee*

Foreword

Comprehensive dental and dental hygiene care is based on the ADPIE model (assessment, diagnosis, planning, implementation, and evaluation). The cornerstone of this model focuses on the information gained through the assessment process, which establishes the direction for the remaining phases of care. The clinician's key function must be to discern normal from abnormal and then thoroughly document and describe those findings. As with previous editions of the *Color Atlas of Common Oral Diseases*, the 4th edition is a valuable aid in the assessment process because it provides a comprehensive overview of normal oral anatomic landmarks, both clinical and radiographic. Additionally, newly redrawn, high-quality diagrams and photographic examples that illustrate basic diagnostic and descriptive terminology are included. Especially notable is a new set of diagrams depicting various histologic changes in the oral tissues.

Once the assessment is complete, the diagnostic phase of care begins. Although the dentist ultimately makes the definitive diagnosis, the dental hygienist plays an integral role gathering data and alerting the dentist to signs of disease or other abnormalities in the oral cavity. The updated edition of this book is an excellent resource to guide the practitioner through the diagnostic process, presenting clinical and radiographic features of the most common diseases found in the oral cavity according to location, color, surface change, and radiographic appearance. In addition, sections are devoted to specific types of lesions, such as dental anomalies, caries, disorders of the gingiva and periodontium, sexually related diseases, and oral conditions affecting infants and children. The presentation of information is well organized, succinct, and accompanied by high-quality photographs, making this atlas extremely useful as a chairside reference and patient-education tool. The latest edition also introduces many photographs not included in prior editions and several new topics such as malocclusion, temporomandibular joint disorders, drug-related oral lesions, and dental resorption.

With the addition of co-author Jill Nield-Gehrig, this textbook has been significantly redesigned to better meet students' needs. Each section now begins with a list of learning objectives geared specifically to both

dental and dental hygiene students. Color-coded sections and highlighted keywords facilitate learning by helping the reader organize information and focus on important facts. The most noteworthy change, the addition of case studies at the end of each section, encourages students to engage in an active learning process by promoting critical thinking and providing an opportunity to apply foundation knowledge to realistic clinical situations. With increased emphasis on case-based testing in Dental and Dental Hygiene National Board examinations, these case studies also provide an additional resource for exam preparation.

This atlas is able to meet the needs of dental and dental hygiene educators who can use it to coordinate lectures with online resources that include an extensive image bank of all clinical and radiographic figures within the book. The images are easily adaptable to PowerPoint slides and provide exquisite examples of typical features of common oral diseases, offering an invaluable resource that may not otherwise be accessible to many educators.

Although primarily focusing on oral pathology, the *Color Atlas of Common Oral Diseases, fourth edition*, will be useful across the curriculum. The comprehensive scope of included topics makes it a beneficial supplement in diverse courses, such as radiology, dental anatomy, head and neck anatomy, and periodontology. Such interdisciplinary utilization encourages collaborative teaching among faculty and, in turn, enhances the learning process.

At its core, the latest edition of this book remains the quintessential *Color Atlas of Common Oral Diseases* yet has evolved into a comprehensive and illustrative resource for dentists, dental hygienists, dental assistants, dental and dental hygiene students, and educators, making it a valuable tool in both the classroom and clinical setting.

PATRICIA A. MCGINLEY, RDH, MS ED
ADJUNCT FACULTY
DEPARTMENTS OF DENTAL HYGIENE
LAKELAND COMMUNITY COLLEGE
KIRTLAND, OHIO
AND
CUYAHOGA COMMUNITY COLLEGE
CLEVELAND, OHIO

Preface

The fourth edition of *Color Atlas of Common Oral Diseases* once again represents several significant evolutionary improvements and expanded coverage as compared with our previous editions. It is with reference to this statement that the co-authors welcome our newest co-author, Jill Nield-Gehrig, a leading author in the dental hygiene field. Readers will see her influence and input on virtually every page of this book.

The most significant changes to be discovered in the fourth edition are perhaps the evolutionary ones encompassing design, content, course director support, and web-based activities. First, a new, more user-friendly and contemporary format has been developed. Each “section” now includes objectives for both dental and dental hygiene students. Sections are now color-coded with tabs at the outer edge of the page margins for easy location of the diseases and disorders. This same color is used to identify the page headers on the left-hand page within each section so readers will easily identify the section they are in. We have also highlighted key words in color throughout the book. These key words help the student focus on important concepts and critical information. The co-authors have also greatly expanded the Glossary at the end of the book and have, where possible, simplified definitions throughout. The text, in addition to being updated with the latest scientific information, has been reviewed extensively to make it more easily read and understood by all levels of students in the various dental school, dental hygiene, and dental assisting programs.

In terms of content, this is one of our most extensive revisions. We have included **nine** new full pages of text and illustrations and **twenty-two** new thought-provoking cases. Thus, the entire book now contains 686 color and radiographic images. The new pages include the following topics: diagnostic and descriptive terminology, occlusion, the temporomandibular joint, resorption, radiopaque/radiolucent lesions, the lamina dura and periodontal membrane space, and drug-related lesions (e.g., “meth mouth” and bisphosphonate osteonecrosis). Throughout the book the authors and editors scrutinized the reproduction quality of the existing images and have provided numerous color corrections. In several cases the images were deemed not up to the authors’ and publishers’ high standards, so these

images have been replaced. New figures were also added by replacing redundant examples and adding new images to illustrate new variations, or adding topics within existing paragraphs of text. For example, instead of two radiographs of supraeruption we now have one radiograph and one clinical image. In another instance, we removed redundant photos of calculus to add illustrative examples of gingival recession. In terms of achieving the highest quality, the authors had all diagrams of the diagnostic and descriptive terminology redrawn to better illustrate the subject matter for the ultimate edification of the student. Several other existing diagrams have also been revised for format improvement and for content corrections. The reader will also note most of the photographs of patients have been layered electronically and placed on a much more pleasing and consistent background.

A major content addition for this edition is the inclusion of case-based application questions that will help students prepare for National Board examinations. To maximize the utility of this new content we consulted with educators and studied the curricular guidelines and released National Board Examinations. This new material involves more than 20 high-quality clinical photos or radiographs or both. These have been placed at the end of each section so that the cases can be reviewed as the material is completed. The clinical cases are provided so students can review the information such that they can (1) describe the lesion, (2) determine if it is normal, variant of normal, or disease, (3) provide a list of other diseases that look similar (i.e., differential diagnosis), (4) provide an assessment work-up plan, and (5) provide a management plan for each case. In order to accommodate the many revisions and yet limit the size of the text, we reformatted the Appendices and renamed them “Clinical Applications and Resources,” with the drug prescription information appearing in a more compressed format.

Support for course directors remains an important focus for the authors and publisher. We understand from listening to the many participants of our Summer Workshops that many institutions and faculty do not have sufficient high-quality oral pathology material, cases, or information on hand to support faculty needs. To meet this formidable need in a truly unprecedented

and “expansive” way, we created a “team of educational and publication experts.” The team includes the three authors, an independent dental hygienist, our editor, and the publisher. Together our team has created and made available electronic PowerPoint lectures for each chapter. who is a highly experienced in this specialized field, has incorporated the appropriate text information into PowerPoint lectures. Individual course directors and instructors who adopt this text can access the complete PowerPoint package free of charge through our publisher’s (Lippincott Williams & Wilkins’) web site, <http://thePoint.lww.com/Langlais4e>. Further, course directors and instructors can add or delete information and illustrations to suit their needs by accessing the Atlas image bank through the same website, <http://thePoint.lww.com/Langlais4e>.

The authors have also incorporated the internet with this book for Continuing Education and resource information. The answers to the Case-Studies will be password-

protected and available on line for the use of course directors who can better challenge their students’ by not including the answers in the book. In addition, multiple reference materials will be listed for quick access by students and faculty alike. As time progresses the authors and publisher will expand the variety of online internet support. We remain committed to providing continuing education opportunities to our readers and teachers through summer workshops, seminars, and presentations at national meetings and locally. *For a current list of locations where we will be presenting the summer workshop, please visit* <http://thePoint.lww.com/Langlais4e>

Also, we encourage you to contact us regarding these opportunities and in ways that we can help you or improve this text.

ROBERT P LANGLAIS, DDS, MS, FACD, FICD

CRAIG S. MILLER, DMD, MS, FACD

JILL NIELD-GEHRIG, RDH, MA

Acknowledgments

We are extremely grateful to our many colleagues who have graciously provided material for publication in this atlas. Without their contributions, this fine quality text would not have been possible. We are also grateful to all practicing dentists and physicians who have referred patients throughout the years to the Department of Dental Diagnostic Science Referral Clinic.

To Jill Nield-Gehrig, who completely reviewed this text, we extend our thanks for her valuable comments and contributions. In addition, we are grateful for the objectives and suggestions she provided for each section.

Marnie Palacios, Christopher McKee, Sam Newman, and David Baker of the Photographic Services Section of Education Resources of The University of Texas Health Science Center receive a special thank you for their masterful job of cropping, background adjustments, and recreating the proper color balance on our illustrations.

We wish to express our appreciation to the following persons for contribution of their clinical photographs and radiographs used in this text:

Dr. Kenneth Abramovitch
Dr. A.M. Abrams
Dr. Marden Alder
Dr. Robert Arm
Dr. Ralph Arnold
Dr. Tom Aufdemorte
Dr. Bill Baker
Dr. Douglas Barnett
Dr. Pete Benson
Dr. Howard Birkholz
Dr. Steve Bricker
Dr. Dale Buller
Dr. James Cecil
Dr. Israel Chilvarquer
Dr. Jerry Cioffi
Dr. Laurie Cohen
Dr. John Coke
Dr. Walter Colon
Dr. Herman Corrales
Dr. James Cottone
Dr. Robert Craig, Jr

Dr. Stephen Dachi
Dr. Douglas Damm
Dr. Maria de Zeuss
Dr. Anna Dongari
Dr. S. Brent Dove
Dr. David Freed
Dr. Franklin Garcia-Godoy
Dr. Birgit Glass
Dr. Tom Glass
Dr. Michael Glick
Dr. Kirsten Gosney
Dr. Ed Heslop
Dr. Michael Huber
Dr. Sheryl Hunter
Dr. Ce.E. Hutter
Dr. J.L. Jensen
Dr. Ron Jorgenson
Dr. Jerald Katz
Dr. George Kaugers
Dr. Gary Klasser
Dr. Tom Kluemper
Dr. Eric Kraus
Dr. Olaf Langland
Dr. Al Lugo
Dr. Curt Lundeen
Dr. Carson Mader
Dr. Nancy Mantich
Dr. Tom McDavid
Dr. John McDowell
Dr. Monique Michaud
Dr. Dale Miles
Dr. John Mink
Dr. David F. Mitchell
Dr. David Molina
Dr. Charles Morris
Dr. Rick Myers
Dr. Stanley Nelson
Dr. Christoffel Nortjé
Dr. Pirkka Nummikoski
Dr. D. Nuggyen
Dr. Linda Otis
Dr. Garnet Pakota
Dr. Joe Petrey

Dr. Roger Rao
Dr. Tom Razmus
Dr. Spencer Redding
Dr. Terry Rees
Dr. Elias Romero
Dr. Michele Saunders
Dr. Stanley Saxe
Dr. Tom Schiff
Dr. James Scuibba
Dr. Jack Sherman
Dr. Sol Silverman
Dr. Larry Skoczylas
Dr. D.B. Smith
Dr. John Tall
Dr. George Taybos
Dr. Geza Terezhalmay
Dr. Mark Thomas
Dr. Nathaniel Triester
Dr. Martin Tyler
Dr. Margot Van Dis
Dr. Michael Vitt
Dr. Elaine Winegard
Dr. Donna Wood
Dr. John Wright
Dr. Jim Zettler

We also wish to express our appreciation to the following educators for attending our Summer Workshops and providing helpful suggestions regarding the format and content of this text:

Dr. William Batson
Dr. Suzanne M. Beatty
Dr. G. David Byers
Michael Campo
Janita D. Cope
Nancy Cuttic

Leslie A. DeLong
Dr. Robert C. Dennison
Dr. Art DiMarco
Kathy Duff
Dr. Robert S. Eldridge
Dr. Marie English
Diana Cooke Gehrke
Deborah Goldstein
Dr. Joel Grand
Jane Gray
Donna Hamil
Rosemary Herman
Dr. Stanley L. Hill
Debbie Hughes
Brenda Knutson
Susan Luethge
Debbie Lyon
Mattie Marcum
Joan McClintock
Patty McGinley
Julie Mettlen
Dr. John A. Olsen
Polly Pope
Carol Robertson
Donna Rollo
Donna Thibodeau
Dana Wood
Mary Ellen Young
Anita Weaver
Karen Sue Williams

Finally, we would like to thank our wives, Denyse and Sherry, for their continuing support throughout this project. Authorship of a high-quality text requires numerous off-duty hours; without the understanding of these two most important people in our lives, this work might never have been completed.

Reviewers

Sharon Barbieri, RDH, MS

Associate Professor
Department of Dental Hygiene
The University of Texas Health Science Center
at San Antonio
San Antonio, Texas

Maria A. Copete, DDS, MS

Associate Professor
College of Dentistry
University of Saskatchewan
Saskatoon SK S7N 5E4 Canada

Richard Foster, DMD

Dental Director
Guilford Technical Community College
Jamestown, North Carolina

Kenneth Horowitz, DMD

Professor
Department of Dental Hygiene
Foothill College
Los Altos Hills, California

Debby Kurtz-Weidinger, RDH, MEd

Assistant Professor
Arizona School of Dentistry and Oral Health
Mesa, Arizona

Beth Monnin, RDH, MSED

Instructor
Department of Dental Hygiene
James A. Rhodes State College
Lima, Ohio

Jill S. Nield-Gehrig, RDH, MA

Allied Health & Public Service Education
Asheville-Buncombe Technical Community College
Asheville, North Carolina

Barbara Ringle, RDH, MEd

Assistant Professor
Department of Dental Hygiene
Cuyahoga Community College
Cleveland, Ohio

Figures Credits

Fig. 1.5	Dr. Kirsten Gosney	Fig. 17.3	Dr. Rick Myers	Fig. 40.3	Dr. Curt Lundeen
Fig. 2.2	Dr. James Cottone	Fig. 17.6	Dr. Ralph Arnold	Fig. 40.5	Dr. James Cottone
Fig. 2.8	Dr. Linda Otis	Fig. 17.8	Dr. Israel Chilvarquer	Fig. 40.6	Dr. Pete Benson
Fig. 3.3	Dr. Kirsten Gosney	Fig. 19.8	Dr. John Mink	Fig. 41.7	Dr. Jack Sherman
Fig. 3.4	Dr. Kirsten Gosney	Fig. 20.3	Dr. Ralph Arnold	Fig. 41.8	Dr. Tom Aufdemorte
Fig. 3.7	Dr. Ralph Arnold	Fig. 20.7	Dr. Geza Terezhalmay	Fig. 42.1	Dr. Kenneth Abramovitch
Fig. 4.1	Dr. Jim Zettler	Fig. 21.3	Dr. Jerry Katz	Fig. 42.3	Dr. James Cottone
Fig. 4.2	Dr. Jim Zettler	Fig. 21.7	Dr. Pirkka Nummikoski	Fig. 42.7	Dr. Anna Dongari
Fig. 4.3	Dr. Jim Zettler	Fig. 21.8	Dr. Pirkka Nummikoski	Fig. 43.3	Dr. Maria de Zeuss
Fig. 4.4	Dr. Tom Kluemper	Fig. 22.2	Dr. Charles Morris	Fig. 43.4	Dr. Stanley Saxe
Fig. 4.5	Dr. Tom Kluemper	Fig. 23.6	Dr. Ralph Arnold	Fig. 43.5	Dr. Ralph Arnold
Fig. 4.6	Dr. Tom Kluemper	Fig. 23.8	Dr. David Molina	Fig. 43.6	Dr. Ralph Arnold
Fig. 4.7	Dr. Joe Petrey	Fig. 24.3	Dr. Birgit Glass	Fig. 44.1	Dr. Monique Michaud
Fig. 4.8	Dr. Joe Petrey	Fig. 24.7	Dr. Terry Rees	Fig. 44.2	Dr. Monique Michaud
Fig. 4.9	Dr. Joe Petrey	Fig. 27.5	Dr. C.E. Hutton	Fig. 44.5	Dr. Roger Rao
Fig. 4.10	Dr. Tom Kluemper	Fig. 27.6	Dr. David F. Mitchell	Fig. 44.6	Dr. Roger Rao
Fig. 4.11	Dr. Tom Kluemper	Fig. 28.7	Dr. Birgit Glass	Fig. 44.7	Dr. Larry Skoczylas
Fig. 4.12	Dr. Tom Kluemper	Fig. 29.3	Dr. Ralph Arnold	Fig. 45.4	Dr. Kenneth Abramovitch
Fig. 7.3	Dr. Stanley Nelson	Fig. 31.1	Dr. Kenneth Abramovitch	Fig. 45.7	Dr. Sol Silverman
Fig. 7.4	Dr. Stanley Nelson	Fig. 31.6	Dr. Garnet Pakota	Fig. 45.8	Dr. Michael Huber
Fig. 7.5	Dr. Stanley Nelson	Fig. 32.3	Dr. Robert Arm	Fig. 46.2	Dr. Bill Baker
Fig. 13.4	Dr. Michael Huber	Fig. 32.8	Dr. Christoffel Nortjé	Fig. 46.8	Dr. Pete Benson
Fig. 13.8	Dr. John Wright	Fig. 34.1	Dr. Israel Chilvarquer	Fig. 47.5	Dr. Jerry Cioffi
Fig. 13.9	Dr. Nancy Mantich	Fig. 34.2	Dr. Elias Romero	Fig. 47.6	Dr. Jerry Cioffi
Fig. 15.1	Dr. Sheryl Hunter	Fig. 34.4	Dr. Pirkka Nummikoski	Fig. 47.7	Dr. Tom Aufdemorte
Fig. 15.2	Dr. Christoffel Nortjé	Fig. 34.10	Dr. D. Nugyyen	Fig. 48.3	Dr. Curt Lundeen
Fig. 15.4	Dr. Ron Jorgenson	Fig. 36.3	Dr. Charles Morris	Fig. 49.1	Dr. Nancy Mantich
Fig. 15.5	Dr. Franklin Garcia-Godoy	Fig. 36.4	Dr. Tom McDavid	Fig. 49.6	Dr. Christoffel Nortjé
Fig. 15.6	Dr. Ron Jorgenson	Fig. 36.5	Dr. Ralph Arnold	Fig. 49.8	Dr. John McDowell
Fig. 15.7	Dr. Ron Jorgenson	Fig. 36.8	Dr. Mark Thomas	Fig. 50.1	Dr. Linda Otis
Fig. 15.8	Dr. Al Lugo			Fig. 50.8	Dr. Geza Terezhalmay
Fig. 15.10	Dr. Barney Olsen				
Fig. 16.6	Dr. Kenneth Abramovitch				

Fig. 52.2	Dr. Dale Miles	Fig. 67.3	Dr. Dale Miles	Fig. 76.6	Dr. Kenneth Abramovitch
Fig. 52.3	Dr. Geza Terezhalmay	Fig. 67.4	Dr. Curt Lundeen	Fig. 77.3	Dr. Donna Wood
Fig. 52.4	Dr. Olaf Langland	Fig. 67.5	Dr. Nancy Mantich	Fig. 77.5	Dr. Tom Schiff
Fig. 52.7	Dr. Dale Miles	Fig. 67.6	Dr. Tom McDavid	Fig. 77.7	Dr. Donna Wood
Fig. 53.1	Dr. Tom McDavid	Fig. 67.7	Dr. Geza Terezhalmay	Fig. 78.5	Dr. Geza Terezhalmay
Fig. 53.2	Dr. Dale Buller	Fig. 67.8	Dr. Michael Vitt	Fig. 78.6	Dr. Tom McDavid
Fig. 53.3	Dr. J.L. Jensen	Fig. 68.6	Dr. Tom McDavid	Fig. 78.7	Dr. Geza Terezhalmay
Fig. 53.4	Dr. S. Brent Dove	Fig. 68.7	Dr. Charles Morris	Fig. 78.8	Dr. Geza Terezhalmay
Fig. 53.5	Dr. James Cottone	Fig. 68.8	Dr. Charles Morris	Fig. 79.1	Dr. Howard Birkholz
Fig. 53.7	Dr. Stephen Dachi	Fig. 69.6	Dr. Tom McDavid	Fig. 79.2	Dr. Michael Huber
Fig. 54.7	Dr. Geza Terezhalmay	Fig. 70.4	Dr. Birgit Glass	Fig. 79.4	Dr. Robert Craig, Jr.
Fig. 56.10	Dr. Michael Vitt	Fig. 70.5	Dr. Jerry Cioffi	Fig. 79.5	Dr. Tom McDavid
Fig. 57.2	Dr. Bill Baker	Fig. 71.1	Dr. Curt Lundeen	Fig. 79.6	Dr. Tom McDavid
Fig. 57.4	Dr. Dale Miles	Fig. 71.2	Dr. Tom McDavid	Fig. 79.7	Dr. Monique Michaud
Fig. 57.8	Dr. Ken Abramovitch	Fig. 72.1	Dr. James Cottone	Fig. 79.8	Dr. Jerry Cioffi
Fig. 59.7	Dr. Spencer Redding	Fig. 72.2	Dr. Linda Otis	Fig. 80.1	Dr. James Cottone
Fig. 59.8	Dr. James Cottone	Fig. 72.3	Dr. Charles Morris	Fig. 80.2	Dr. Marden Alder
Fig. 60.5	Dr. Linda Otis	Fig. 73.5	Dr. Birgit Glass and Dr. Tom Glass	Fig. 80.3	Dr. James Cottone
Fig. 60.6	Dr. Es Heslop	Fig. 73.6	Dr. Birgit Glass and Dr. Tom Glass	Fig. 80.4	Dr. James Cottone
Fig. 60.7	Dr. Tom Razmus	Fig. 73.7	Dr. Birgit Glass and Dr. Tom Glass	Fig. 80.5	Dr. Geza Terezhalmay
Fig. 61.3	Dr. Magot Van Dis	Fig. 73.8	Dr. Birgit Glass and Dr. Tom Glass	Fig. 80.6	Dr. Geza Terezhalmay
Fig. 61.4	Dr. Magot Van Dis	Fig. 74.1	Dr. Michelle Saunders	Fig. 80.7	Dr. Laurie Cohen and Dr. John Coke
Fig. 61.5	Dr. Magot Van Dis	Fig. 74.2	Dr. Carson Mader	Fig. 81.3A	Dr. Walter Colon
Fig. 61.6	Dr. Larry Skoczylas	Fig. 74.8	Dr. Steve Bricker	Fig. 81.4	Dr. Michael Vitt
Fig. 62.3	Dr. Robert Craig, Jr.	Fig. 75.1	Dr. Sol Silverman	Fig. 81.7	Dr. Ed Heslop
Fig. 62.4	Dr. Robert Craig, Jr.	Fig. 75.2	Dr. Tom McDavid	Fig. 81.8	Dr. Ed Heslop
Fig. 63.2	Dr. Birgit Glass	Fig. 75.4	Dr. Tom McDavid	Fig. 82.1	Dr. Jerry Cioffi
Fig. 63.5	Dr. Tom Razmus	Fig. 75.5	Dr. Tom McDavid	Fig. 82.3	Dr. Michael Huber
Fig. 64.2	Dr. James Cottone	Fig. 75.7	Dr. Terry Rees	Fig. 82.4	Dr. Michael Glick
Fig. 64.3	Dr. James Cottone	Fig. 75.8	Dr. Eric Kraus and Dr. Herman Corrales	Fig. 82.7	Dr. Michael Huber
Fig. 65.2	Dr. Geza Terezhalmay	Fig. 76.1	Dr. Eric Kraus	Fig. 82.8	Dr. George Kaugers
Fig. 65.6	Dr. James Cottone	Fig. 76.3	Dr. Tom McDavid and Dr. Martin Tyler	Fig. 83.1	Dr. James Cecil and Dr. Douglas Damm
Fig. 65.8	Dr. Ken Abramovitch	Fig. 76.4	Dr. Tom McDavid and Dr. Martin Tyler	Fig. 83.2	Dr. Gary Klasser
Fig. 66.2	Dr. David Freed			Fig. 83.3	Dr. Nathaniel Triester
Fig. 66.3	Dr. Linda Otis			Fig. 83.4	Dr. Nathaniel Triester
Fig. 66.4	Dr. Linda Otis			Fig. 83.5	Dr. James Scuibba
				Fig. 83.6	Dr. James Scuibba
				Fig. 83.7	Dr. James Scuibba
				Fig. 83.8	Dr. George Taybos
				Fig. 83.9	Dr. Walton Colon

Contents

SECTION 1. Anatomic Landmarks	1	DIAGNOSTIC AND DESCRIPTIVE TERMINOLOGY	
LANDMARKS OF THE ORAL CAVITY		Wheal, Scar, Fissure, Sinus	20
Lips, Labial and Buccal Mucosa, Parotid Papilla, Floor of the Mouth, Hard Palate, Soft Palate, Oropharynx and Tonsils	2	DIAGNOSTIC AND DESCRIPTIVE TERMINOLOGY	
LANDMARKS OF THE TONGUE AND VARIANTS OF NORMAL		Papule, Plaque, Nodule, Tumor	22
Normal Tongue Anatomy, Fissured Tongue (Plicated Tongue, Scrotal Tongue), Ankyloglossia, Lingual Varicosity (Phlebectasia)	4	DIAGNOSTIC AND DESCRIPTIVE TERMINOLOGY	
LANDMARKS OF THE PERIODONTIUM		Vesicle, Pustule, Bulla, Cyst	24
Periodontium, Alveolar Mucosa and Frenal Attachments, Mucogingival Junction, Attached Gingiva and Free Marginal Gingiva	6	DIAGNOSTIC AND DESCRIPTIVE TERMINOLOGY	
OCCCLUSION AND MALOCCLUSION		Normal, Hypotrophy and Atrophy, Hypertrophy, Hypoplasia	26
Class I Occlusion, Class II Occlusion, Class III Occlusion	8	DIAGNOSTIC AND DESCRIPTIVE TERMINOLOGY	
RADIOGRAPHIC LANDMARKS: MAXILLA		Hyperplasia, Metaplasia, Dysplasia, Carcinoma	28
Anterior Midline Region, Anterior Lateral Region, Canine Region, Premolar Region, Molar Region, Tuberosity Region	10	SECTION 2 CASES	30
RADIOGRAPHIC LANDMARKS: MANDIBLE		SECTION 3. Oral Conditions Affecting Infants and Children	31
Incisor-Canine Region, Premolar and Molar Regions, Premolar Region, Buccal Aspect Molar Region, Lingual Aspect Molar Region, Internal Aspect Molar Region	12	ORAL CONDITIONS AFFECTING INFANTS AND CHILDREN	
TEMPOROMANDIBULAR JOINT		Commisural Lip Pits, Paramedian Lip Pits, Cleft Lip, Cleft Palate, Bifid uvula	32
Normal Anatomy, Normal Opening, Deviation on Opening, Posterior Open Bite–Midline Deviation– Crossbite, Anterior Open Bite, Crossbite	14	ORAL CONDITIONS AFFECTING INFANTS AND CHILDREN	
SECTION 1 CASES	16	Congenital Epulis, Melanotic Neuroectodermal Tumor of Infancy, Dental Lamina Cysts, Natal Teeth, Eruption Cyst (Gingival Eruption Cyst, Eruption Hematoma), Congenital Lymphangioma, Thrush (Candidiasis, Moniliasis), Parulis (Gum Boil)	34
SECTION 2. Diagnostic and Descriptive Terminology	17	SECTION 3 CASES	36
DIAGNOSTIC AND DESCRIPTIVE TERMINOLOGY		SECTION 4. Dental Anomalies	37
Macule, Patch, Erosion, Ulcer	18	ALTERATIONS IN TOOTH MORPHOLOGY	
		Microdontia, Macrodontia, Dens Invaginatus (Dens in Dente) Cusp of Carabelli, Dens Evaginatus (Leong's Tubercle), Protostylid, Talon Cusp	38

ALTERATIONS IN TOOTH MORPHOLOGY

Introduction, Fusion, Gemination, Twinning,
Concrescence, Palato-Gingival Groove 40

ALTERATIONS IN TOOTH MORPHOLOGY

Supernumerary Roots, Ectopic Enamel: Enamel
Pearl, Ectopic Enamel: Cervical Enamel Extensions,
Dilaceration, Bulbous Root, Hypercementosis,
Taurodontism, Shovel-Shaped Incisor Syndrome 42

ALTERATIONS IN TOOTH NUMBERS: HYPODONTIA

Hypodontia, Acquired Hypodontia, Ankylosis,
Ectodermal Dysplasia 44

ALTERATIONS IN TOOTH NUMBERS: HYPERDONTIA

Hyperdontia, Cleidocranial Dysplasia, Gardner
Syndrome 46

ALTERATIONS IN TOOTH STRUCTURE AND COLOR

Enamel Hypoplasia, Enamel Hypoplasia:
Environmental Types, Turner's Tooth, Fluorosis
or Mottled Enamel, Amelogenesis Imperfecta 48

ALTERATIONS IN TOOTH STRUCTURE AND COLOR

Dentinogenesis Imperfecta (Hereditary Opalescent
Dentin) and Dentin Dysplasia, Regional
Odontodysplasia (Ghost Teeth) 50

ALTERATIONS IN TOOTH COLOR

Intrinsic Discoloration (Staining), Nonvital Teeth,
Tetracycline Staining, Fluorosis, Extrinsic
Staining 52

TOOTH ERUPTION AND ALTERATION IN TOOTH POSITION

Rotated Teeth, Axial Tilting, Ectopic Eruption,
Orthodontic Tooth Movement, Transposition,
Translocation, Distal Drift, Migration, Partial
(Delayed) Eruption, Supraeruption (Extrusion) 54

ACQUIRED DEFECTS OF TEETH: NONCARIOUS LOSS OF TOOTH STRUCTURE

Attrition, Abrasion, Abfraction, Erosion 56

ALTERATION IN ROOT STRUCTURE

Resorption, False Resorption, External Resorption,
Eruption-Related External Root Resorption,
Inflammatory External Resorption 58

ALTERATION IN ROOT STRUCTURE

Orthodontic External Root Resorption, External
Root Resorption in Reimplanted and Transplanted
Teeth, External Coronal Resorption, Cervical
Resorption, Multiple Cervical Resorption of
Hyperparathyroidism, Coronal Internal
Resorption, Inflammatory Internal Root
Resorption, Replacement/Metaplastic
Internal Root Resorption 60

SECTION 4 CASES

62

SECTION 5. Dental Caries

63

DENTAL CARIES

Class I Caries, Class II Caries, Class III Caries 64

DENTAL CARIES

Class IV Caries, Class V Caries, Class VI Caries,
Root Caries, Recurrent Caries (Secondary
Caries) 66

DENTAL CARIES AND SEQUELAE

Caries Progression, Pulp Polyp, Periapical
Inflammation (Apical Periodontitis), Periapical
Abscess 68

SECTION 5 CASES

70

SECTION 6. Radiolucent and Radiopaque Lesions of the Jaws

71

RADIOLUCENT LESIONS OF THE JAWS: CYSTS

Cysts of the Jaws, Nasopalatine Duct Cyst (Incisive
Canal Cyst) Lateral Periodontal Cyst, Botryoid
Lateral Periodontal Cyst, Dentigerous Cyst,
Odontogenic Keratocyst (OKC), Nevroid Basal Cell
Carcinoma Syndrome (Gorlin-Goltz Syndrome),
Paradental Cyst (Buccal Bifurcation Cyst), Simple
(Traumatic) Bone Cyst 72

RADIOLUCENT LESIONS OF THE JAWS: TUMORS

Unicystic (Mural) Ameloblastoma, Adenomatoid
Odontogenic Tumor, Calcifying Epithelial
Odontogenic (Pindborg) Tumor, Ameloblastic Fibro-
Odontoma, Odontoameloblastoma (Ameloblastic
Odontoma), Ameloblastoma, Myxoma, Central
Giant Cell Granuloma 74

RADIOPAQUE LESIONS OF THE JAWS: BONY LESIONS

Exostosis, Mandibular Tori, Palatal Tori, Osteoma,
Reactive Subpontine (Hyperostosis) Exostosis 76

PERIAPICAL RADIOPAACITIES

Socket Sclerosis, Idiopathic Osteosclerosis
(Enostosis), Condensing Osteitis 76

RADIOPAQUE-RADIOLUCENT LESIONS OF THE JAWS

Odontoma, Compound Odontoma, Complex
Odontoma, Cemento-Osseous Dysplasias,
Periapical Cemento-Osseous Dysplasia,
(Periapical Cemental Dysplasia, Cementoma),
Focal Cemento-Osseous Dysplasia, Florid Cemento-
Osseous Dysplasia, Cementoblastoma,
Ossifying Fibroma 78

SECTION 6 CASES

80

SECTION 7. Disorders of Gingiva and Periodontium

81

PERIODONTAL DISEASES: PLAQUE, CALCULUS, AND REGRESSIVE CHANGES

Plaque, Calculus, Gingival Recession, Dehiscence and Fenestration 82

GINGIVITIS

Gingivitis, Gingivitis Caused by Mouth Breathing, Necrotizing Ulcerative Gingivitis, Actinomycotic Gingivitis, Focal Eruption Gingivitis, Prophy Paste (Foreign-body) Gingivitis 84

PERIODONTITIS

Periodontitis, Mild Periodontitis, Moderate Periodontitis, Advanced Periodontitis, Periodontal Abscess 86

RADIOGRAPHIC FEATURES OF PERIODONTAL DISEASE

Local Factors: Overhang, Local Factors: Open Contact and Poor Restoration Contour, Bone Loss: Localized, Bone Loss: Generalized, Bone (Infrabony), One-Wall Infrabony Defect, Two-Wall Infrabony Defect, Three-Wall Infrabony Defect and Moat Defect 88

RADIOGRAPHIC ALTERATIONS OF PERIODONTAL LIGAMENT AND LAMINA DURA

Periodontal Ligament Space and Lamina Dura, Acute Apical Periodontitis, Periodontitis, Traumatic Occlusion and Orthodontic Tooth Movement, Scleroderma, Malignant Disease-Related Periodontal Defect, Ankylosis, Fibrous Dysplasia, Paget's Disease and Hyperparathyroidism 90

LOCALIZED GINGIVAL LESIONS

Pyogenic Granuloma, Peripheral Giant Cell Granuloma, Peripheral Ossifying Fibroma, Irritation Fibroma, Peripheral Odontogenic Fibroma, Desmoplastic Fibroma 92

LOCALIZED GINGIVAL LESIONS

Parulis (Gumboil), Pericoronitis (Operculitis), Periodontal Abscess, Epulis Fissuratum, Gingival Carcinoma 94

GENERALIZED GINGIVAL ENLARGEMENTS

Fibromatosis Gingivae, Drug-Induced Gingival Overgrowth, Primary Herpetic Gingivostomatitis 96

ENDOCRINE-ASSOCIATED GINGIVAL ENLARGEMENTS

Hormonal Gingivitis (Pregnancy Gingivitis), Diabetic Gingivitis, Gingival Edema of Hypothyroidism 98

SPONTANEOUS GINGIVAL BLEEDING

Leukemic Gingivitis, Agranulocytosis (Neutropenia), Cyclic Neutropenia, Thrombocytopathic and Thrombocytopenic Purpura 100

SECTION 7 CASES

102

SECTION 8. Abnormalities by Location

103

CONDITIONS PECULIAR TO THE TONGUE

Scalloped Tongue (Crenated Tongue), Macroglossia, Hairy Tongue (Lingua Villosa, Coated Tongue), Hairy Leukoplakia 104

CONDITIONS PECULIAR TO THE TONGUE

Geographic Tongue (Benign Migratory Glossitis, Erythema Migrans), Geographic Stomatitis (Acreta Erythema Migrans), Anemia, Fissured Tongue 106

CONDITIONS PECULIAR TO THE TONGUE

Cyst of Blandin-Nuhn (Lingual Mucus-Retention Phenomenon), Median Rhomboid Glossitis, Granular Cell Tumor, Lingual Thyroid, Body Piercing (Oral Jewelry) 108

CONDITIONS PECULIAR TO THE LIP

Actinic Cheilosis (Actinic Cheilitis), Candidal Cheilitis, Angular Cheilitis (Perléche), Exfoliative Cheilitis 110

NODULES OF THE LIP

Mucocele (Mucous Extravasation Phenomena), Accessory Salivary Gland Tumor, Nasolabial Cyst (Nasoalveolar Cyst), Implantation Cyst (Epithelial Inclusion Cyst), Mesenchymal Nodules and Tumors 112

SWELLINGS OF THE LIP

Angioedema, Cheilitis Glandularis, Orofacial Granulomatosis (Cheilitis Granulomatosa), Trauma, Cellulitis 114

SWELLINGS OF THE FLOOR OF THE MOUTH

Dermoid Cyst, Ranula (Mucocele of the Sublingual Gland), Salivary Duct Cyst, Salivary Calculi (Sialoliths), Mucocele (Mucus-Retention Phenomenon) 116

SWELLINGS OF THE PALATE

Palatal Torus (Torus Palatinus), Lipoma, Nasopalatine Duct Cyst (Incisive Canal Cyst), Periapical Abscess, Periodontal Abscess, Lymphoid Hyperplasia, Lymphoma 118

SWELLINGS OF THE PALATE: SALIVARY GLAND LESIONS

Sialadenitis, Necrotizing Sialometaplasia, Benign Accessory Salivary Gland Neoplasm, Malignant Accessory Salivary Gland Neoplasm

120

SWELLINGS OF THE FACE

Odontogenic Infection, Buccal Space Infection, Masseteric (Masticator) Space Infection, Infraorbital Space Infection, Ludwig Angina

122

SWELLINGS OF THE FACE

Sialadenosis, Warthin Tumor (Papillary Cystadenoma Lymphomatosum), Sjögren Syndrome, Cushing Disease and Syndrome, Masseter Hypertrophy, Neurofibromatosis (von Recklinghausen Disease), Cystic (Lymphangioma), Hygroma, Ewing Sarcoma

124

CONDITIONS PECULIAR TO THE FACE

Angioedema, Emphysema, Postoperative Bleeding, Bell Palsy

126

SECTION 8 CASES

128

SECTION 9. Intraoral Findings by Color Changes 129**WHITE LESIONS**

Fordyce Granules, Linea Alba, Leukoedema, Morsicatio Buccarum

130

WHITE LESIONS

White Sponge Nevus (Familial White Folded Dysplasia), Traumatic White Lesions, Leukoplakia

132

TOBACCO-ASSOCIATED WHITE LESIONS

Cigarette Keratosis, Nicotine Stomatitis (Smoker's Palate), Snuff Dipper's Patch (Tobacco Chewer's Lesion, Snuff Keratosis), Verrucous Carcinoma (of Ackerman)

134

RED LESIONS

Purpura (Petechiae, Ecchymoses, Hematoma), Varicosity (Varix), Thrombus, Hemangioma

136

RED LESIONS

Hereditary Hemorrhagic Telangiectasia (Osler-Weber-Rendu Syndrome), Sturge-Weber Angiomatosis (Sturge-Weber or Encephalotrigeminal Syndrome)

138

RED AND RED-WHITE LESIONS

Erythroplakia, Erythroleukoplakia and Speckled Erythroplakia, Squamous Cell Carcinoma

140

RED AND RED-WHITE LESIONS

Lichen Planus, Lichenoid Mucositis (Lichenoid Drug Reaction/Eruption)

142

RED AND RED-WHITE LESIONS

Lupus Erythematosus, Lichenoid and Lupuslike Drug Eruption

144

RED AND RED-WHITE LESIONS

Pseudomembranous Candidiasis (Thrush), Chronic Hyperplastic Candidiasis, Erythematous Candidiasis, Acute Atrophic Candidiasis (Antibiotic Sore Mouth), Angular Cheilitis, Chronic Atrophic Candidiasis (Denture Stomatitis)

146

PIGMENTED LESIONS

Melanoplakia (Physiologic Pigmentation), Tattoo, Ephelis (Freckle), Smoker's Melanosis (Tobacco-Associated Pigmentation)

148

PIGMENTED LESIONS

Oral Melanotic Macule (Focal Melanosis), Nevus, Melanoma

150

PIGMENTED LESIONS

Peutz-Jeghers Syndrome (Hereditary Intestinal Polyposis), Addison Disease (Adrenal Cortical Insufficiency), Heavy Metal Pigmentation

152

SECTION 9 CASES

154

SECTION 10. Intraoral Findings by Surface Change 155**NODULES**

Retrocuspid Papilla, Oral Lymphoepithelial Cyst, Torus, Exostosis, and Osteoma

156

NODULES

Irritation Fibroma, Peripheral Odontogenic Fibroma, Giant Cell Fibroma, Lipoma, Fibrolipoma, Traumatic Neuroma, Neurofibroma

158

PAPULONODULES

Oral Squamous Papilloma (Papilloma), Verruca Vulgaris, Focal Epithelial Hyperplasia (Heck Disease), Condyloma Acuminatum (Venereal Wart), Lymphangioma

160

VESICULOBULLOUS LESIONS

Primary Herpetic Gingivostomatitis, Recurrent Herpes Simplex Infection, Herpangina

162

VESICULOBULLOUS LESIONS

Varicella (Chickenpox), Herpes Zoster (Shingles), Hand-Foot-and-Mouth Disease

164

VESICULOBULLOUS LESIONS

Allergic Reactions, Localized Anaphylaxis,
Generalized Anaphylaxis, Allergic Stomatitis,
Angioedema, Delayed Hypersensitivity, Contact
Stomatitis, Plasma Cell Gingivitis 166

VESICULOBULLOUS LESIONS

Erythema Multiforme, Oral Erythema Multiforme,
Erythema Multiforme, Stevens-Johnson Syndrome
(Erythema Multiforme Major), Toxic Epidermal
Necrolysis 168

VESICULOBULLOUS LESIONS

Pemphigus Vulgaris, Pemphigoid (Bullous and
Cicatricial [Benign Mucous Membrane]) 170

ULCERATIVE LESIONS

Traumatic Ulcer, Recurrent Aphthous Stomatitis
(Minor Aphthae, Aphthous Ulcer),
Pseudoaphthous 172

ULCERATIVE LESIONS

Major Aphthous, Herpetiform Ulceration, Behçet
Syndrome (Oculo-Oral-Genital Syndrome) 174

ULCERATIVE LESIONS

Granulomatous Ulcer, Squamous Cell Carcinoma,
Chemotherapeutic Ulcer 176

SECTION 10 CASES

178

**SECTION 11. Oral Manifestations of Sexual
Conditions and Systemic Drug Therapies**

179

SEXUALLY RELATED AND SEXUALLY TRANSMISSIBLE CONDITIONS

Traumatic Conditions, Sexually Transmitted
Pharyngitis, Infectious Mononucleosis,
Syphilis 180

HIV INFECTION AND AIDS

Acquired Immunodeficiency Syndrome (AIDS),
Oral Bacterial Infections, Necrotizing Periodontal
Diseases, Necrotizing Ulcerative Gingivitis,
Necrotizing Ulcerative Periodontitis (NUP), Oral
Fungal Infections, Linear Gingival Erythema,
Pseudomembranous Candidiasis 182

HIV INFECTION AND AIDS

Oral Viral Infections, Varicella-Zoster Virus,
Cytomegalovirus (CMV), Epstein-Barr Virus (EBV)
and Hairy Leukoplakia, Human Papillomavirus

(HPV), Condyloma Acuminatum, Oral Malignancies:
Non-Hodgkin Lymphoma and Squamous Cell
Carcinoma 184

ORAL EFFECTS OF DRUGS AND THERAPIES

Meth Mouth, Graft-Versus-Host
Disease, Bisphosphonate Osteonecrosis,
Drug-Induced Hyperpigmentation 186

SECTION 11 CASES

188

SECTION 12. Clinical Applications and Resources

189

RX ABBREVIATIONS

190

PRESCRIPTIONS AND THERAPEUTIC PROTOCOLS

191

ANTIBIOTIC PROPHYLAXIS

192

ANTIBIOTIC THERAPY

193

ANTIMICROBIAL TOPICAL AGENTS AND RINSES

194

ANTIFUNGAL THERAPY

195

ANTIVIRAL THERAPY

196

TOPICAL ORAL ANESTHETIC AGENTS

198

ANTI-ANXIETY AGENTS

199

FLUORIDE THERAPY

200

MUCOSAL ULCER MEDICATIONS

201

NUTRIENT DEFICIENCY THERAPY

203

SALIVA SUBSTITUTE

204

SEDATIVE/HYPNOTICS

205

TOBACCO CESSATION

206

**GUIDE TO DIAGNOSIS AND MANAGEMENT OF
COMMON ORAL LESIONS**

207

GLOSSARY

227

INDEX

241

SECTION 1

Anatomic Landmarks

Dental and Dental Hygiene Objectives:

- Recognize, define, and describe the soft tissue structures and landmarks of the anterior and posterior oral cavity.
- Recognize, define, and describe the soft tissue structures and landmarks of the floor of the mouth, tongue, and palate.
- Recognize, define, and describe the soft tissue structures and landmarks of the periodontium.
- Recognize, define, and describe the bony structures and landmarks of the maxilla and mandible and adjacent regions.
- Recognize, define, and describe common variants of normal.
- In the clinical setting, identify intraoral soft tissue structures and anatomic landmarks in a patient's mouth.

LANDMARKS OF THE ORAL CAVITY

Lips (Fig. 1.1) The lips form the outer border of the oral cavity. They are covered by mucosa and a surface layer of parakeratin. Beneath this is connective tissue and rich blood supply. Deeper are muscles that control lip movement (orbicularis oris, levator, and depressor oris). Lips appear pink-red but can vary in color depending on the pigmentation of the patient, sun exposure, and history of trauma. The junction of the lips with the labial mucosa is the **wet line**, the point of contact of the upper and lower lips. The **vermilion** is the portion external to the wet line. The **vermilion border** is the junction of the lip with the skin. The lips should be visually inspected and palpated by everting during the oral examination. The surface should be smooth and uniform in color; the border should be smooth and well delineated.

Labial and Buccal Mucosa (Figs. 1.2 and 1.3) The **labial mucosa** is the inside lining of the lips; the **buccal mucosa** is the inner lining of the cheeks. Each is covered by thin, pink parakeratotic epithelium. The mucosa is usually pink or brownish-pink with small red capillaries nourishing the region. Minor **salivary gland ducts** empty onto the surface of the mucosa. The surface of the labial mucosa is covered by small orifices that emit mucinous saliva. The buccal mucosa is the inner aspect of the cheeks. It broadens bilaterally from the labial mucosa to the retro-molar pad and pterygomandibular raphe. Deposits of fat within the buccal connective tissue can make it appear yellow or tan. Accessory salivary glands are present in this region and moisten the oral mucosa. The **caliculus angularis** is a normal pinkish papule located buccally at the commissure.

Parotid Papilla (Fig. 1.4) The **parotid papilla** is a triangular, raised, pink papule on the buccal mucosa adjacent to the maxillary first molars bilaterally. The papilla forms the end of **Stensen's duct**, the excretory duct of the parotid gland. The gland is milked by drying the papilla with gauze, pressing the fingers below the mandible, and extending pressure upward and over the gland. In health, clear saliva should flow from the duct.

Floor of the Mouth (Fig. 1.5) The **floor of the mouth** is the region below the front, anterior half of the tongue. It is composed of thin, pink parakeratinized epithelium, connective tissue, salivary glands, and associated nerves and blood vessels. The floor of the mouth has U-shaped boundaries bordered anterolaterally by the dental arch and posteriorly by the ventral tongue surface. The anterior portion is smooth, uniform, and covered by mucosa. The **lingual frenum** is located along the midline of the posterior portion. Between the two halves is an elevated area under which **Wharton's duct** of the submandibular gland lies. Saliva from the submandibular gland exits through an elevated papule called the **sublingual caruncle** to moisten the floor of the mouth. Along the posterior portion of the caruncle

are multiple small openings, the "**ducts of Rivinus**," that carry saliva from the sublingual salivary gland. Beneath these structures lies a pair of **mylohyoid muscles** that function in lifting the tongue and hyoid bone.

Hard Palate (Fig. 1.6) The **hard palate** forms the roof of the oral cavity. It is composed of squamous epithelium, connective tissue, minor salivary glands and ducts (in the posterior two thirds only), **periosteum**, and the palatine processes of the maxilla. Anatomically, it consists of several structures. The **incisive papilla** is directly behind and between the maxillary incisors. It is a raised, pink ovoid structure that overlies the nasopalatine foramen. The **rugae** are fibrous ridges that are located slightly posterior to the incisive papilla, in the anterior third of the palate. They run laterally from the midline to within several millimeters of the attached gingiva of the anterior teeth. A little further back are the **lateral vaults**, alveolar bones that support the palatal aspects of the posterior teeth. In the center of the hard palate is the **median palatal raphe**, a yellow-white fibrous band that appears at the junction of the right and left palatine processes.

Soft Palate (Fig. 1.7) The **soft palate** is located posterior to the hard palate. It is unique from the hard palate in that the soft palate lacks bony support and has more minor salivary glands and lymphoid and fatty tissue than the hard palate. The soft palate functions during mastication and swallowing. It is elevated during swallowing by the levator palati and tensor palati muscles, and motor innervated by cranial nerves IX and X. The **median palatal raphe** is more prominent and thicker in the soft palate. Just lateral to the raphe are the **fovea palatinae**. The fovea are 2-mm excretory ducts of minor salivary glands. They are landmarks of the junction between the hard and soft palates. At the midline and distal aspect of the soft palate is the **uvula**.

Oropharynx and Tonsils (Fig. 1.8) The **oropharynx** is the junction between the mouth and the esophagus. The borders of the oropharynx are the uvula along the anterior aspect, the two tonsillar pillars (fauces) along the anterolateral aspect, and the pharyngeal wall at the posterior aspect. The tonsils are lymphoid tissue located within two pillars. The anterior tonsillar pillar is formed by the palatoglossus muscle that runs downward, outward, and forward to the base of the tongue. The posterior pillar is larger and runs posteriorly. It is formed by the palatopharyngeus muscle. The **tonsils** are dome-shaped soft-tissue structures that have surface crypts and invaginations (folds), which serve to capture invading microbes. Tonsils enlarge during adolescence (a lymphoid growth period) and during infectious, inflammatory, and neoplastic processes. Islands of tonsillar tissue are seen on the surface of the posterior pharyngeal wall. **Waldeyer's ring** is the ring of adenoid tissue formed by the tonsillar tissue found on the posterior tongue (lingual tonsils), pharynx (pharyngeal tonsils), and fauces (tonsillar pillars).



Fig. 11. Lips: normal, healthy appearance.



Fig. 12. Labial mucosa: inner lining of the lips.



Fig. 13. Buccal mucosa: and calculus angularis.



Fig. 14. Parotid papilla: adjacent to maxillary first molar.



Fig. 15. Floor of mouth: with central lingual frenum.



Fig. 16. Hard palate: incisive papilla and rugae in anterior third.



Fig. 17. Soft palate: fovea palatinae and median palatal raphe.



Fig. 18. Oropharynx and tonsillar pillars.

LANDMARKS OF THE TONGUE AND VARIANTS OF NORMAL

Normal Tongue Anatomy (Figs. 2.1–2.5) The tongue is a compact organ composed of skeletal muscles that has important functions in taste, chewing, swallowing (deglutition), and speech. The **dorsum** (upper surface) of the tongue is covered by a protective layer of stratified squamous epithelium and numerous mucosal projections that form papillae. Four types of papillae cover the dorsum of the tongue: filiform, fungiform, circumvallate, and foliate papillae. **Filiform papillae** are the smallest but the most numerous. They are slender, hair-like, cornified stalks that serve to protect the tongue. They appear pink in patients with good oral hygiene, but may be red or white, if irritated or inflamed. **Fungiform papillae** are noncornified, round, mushroom-shaped papillae found interspersed between, and slightly elevated above the filiform papillae. They are brighter red, broader in width, and fewer in number (~300 to 500), than the filiform papillae. Each fungiform papilla contains two to four taste buds that confer the ability to taste (salty, sweet, sour, and bitter). Fungiform papillae are most numerous on the lateral border and anterior tip of the tongue, and can be stained with blue food color and viewed. Fungiform papillae sometimes contain brown pigmentation, especially in melanoderms.

The largest papillae, the **circumvallate papillae**, also contain taste buds. There are 8 to 12 circumvallate papillae arranged in a V-shaped row at the posterior aspect of the dorsum of the tongue. They appear as 2- to 4-mm pink elevations surrounded by a narrow trench, the **sulcus terminalis**.

Careful examination of the lateral border of the posterior region of the tongue reveals the **foliate papillae**. These papillae are leaflike projections oriented as vertical folds. Foliate papillae are more prominent in children and young adults than in older adults. Corrugated hypertrophic lymphoid tissue (**lingual tonsil**) extending into this area from the posterior dorsal root of the tongue may sometimes be mistakenly called foliate papillae.

On the ventral surface (underside) of the tongue are linear projections known as the **plica fimbriata**. The plica fimbriata have little known function in humans, but contain taste buds in newborns and other primates. Occasionally the fimbriata are brown in dark-skinned individuals.

Fissured Tongue (Plicated Tongue, Scrotal Tongue) (Fig. 2.6) **Fissured tongue** is a variation of normal tongue anatomy that consists of a single midline fissure, double fissures, or multiple fissures of the anterior two thirds of the dorsal surface of the tongue. Various patterns, lengths, and depths of the fissures have been observed. The cause is often unknown, but it often develops with

increasing age and in patients who have xerostomia (dry mouth). About 1% to 5% of the population is affected. The frequency of the condition is equal in men and women. It occurs commonly in patients with Down syndrome and in combination with geographic tongue. Fissured tongue is a component of the **Melkersson-Rosenthal syndrome** (fissured tongue, cheilitis granulomatosa, and unilateral facial nerve paralysis).

Tongue fissures may become secondarily inflamed and cause halitosis as a result of food impaction; thus, brushing the tongue to keep the fissures clean is recommended. The condition is benign and does not cause pain.

Ankyloglossia (Fig. 2.7) The **lingual frenum** is normally attached to the ventral tongue and genial tubercles of the mandible. If the frenum fails to attach properly to the tongue and genial tubercles, but instead fuses to the floor of the mouth or lingual gingiva and the ventral tip of the tongue, the condition is called **ankyloglossia** or “tongue-tie.” This congenital condition is characterized by (i) an abnormally short, malpositioned, and thickened lingual frenum and (ii) a tongue that cannot be extended or retracted. The fusion may be partial or complete. Partial fusion is more common. If the condition is severe, speech may be affected. Surgical correction and speech therapy are necessary if speech is defective or if a mandibular denture or removable partial denture is planned. The estimated frequency of ankyloglossia is one case per 1,000 births.

Lingual Varicosity (Phlebectasia) (Fig. 2.8) **Lingual varicosities**, enlarged dilated veins on the ventral surface of the tongue, are a common finding in elderly adults. The cause of these vascular dilatations is either a blockage of the vein by an internal foreign body, such as an atherosclerotic plaque, or the loss of elasticity of the vascular wall as a result of aging. Intraoral varicosities most commonly appear superficially on the ventral surface of the anterior two thirds of the tongue and may extend onto the lateral border and floor of the mouth. Men and women are affected equally.

Varicosities appear as red-blue to purple fluctuant papules or nodules. Individual varices may be prominent and tortuous or small and punctate. Palpation does not elicit pain but can move the blood temporarily out of the vessel, thereby flattening the surface appearance. **Diascopy** (pressing against the lesion with a clear plastic tube or glass slide) causes varices to blanch. When many lingual veins are prominent, the condition is called “**phlebectasia linguae**” or “**caviar tongue**.” The lip and labial commissure are other frequent sites of phlebectasia. Treatment of this condition is not required, unless for cosmetic reasons.



Fig. 2.1. Filiform and fungiform papillae of tongue.

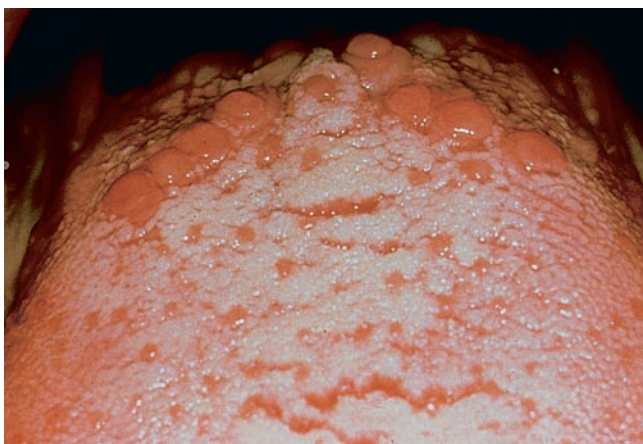


Fig. 2.2. Circumvallate papillae forming a V-shaped row.

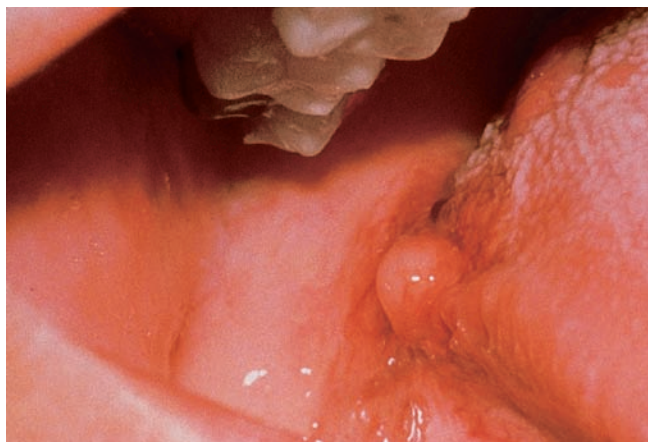


Fig. 2.3. Foliate papilla: posterolateral aspect of tongue.



Fig. 2.4. Lingual tonsil at dorsolateral aspect of tongue.



Fig. 2.5. Plica fimbriata in person who is pigmented.



Fig. 2.6. Fissured tongue: dorsal aspect.



Fig. 2.7. Ankyloglossia: not causing a speech impediment.



Fig. 2.8. Lingual varicosities: on ventral tongue.

LANDMARKS OF THE PERIODONTIUM

Periodontium (Figs. 3.1 and 3.2) The periodontium is the tissue that immediately surrounds and supports the teeth. It consists of alveolar bone, periosteum, periodontal ligament, gingival sulcus, and gingiva; each of these components contributes to stabilizing the tooth within the jaws. The **alveolar bone** is composed of cancellous or spongy bone. It is located between the cortical plates and is penetrated by blood vessels and marrow spaces. The **periosteum** is the dense connective tissue attached to and covering the outer surface of the alveolar bone. Teeth are anchored to alveolar bone by the periodontal ligament that attaches to the cementum that covers the roots of teeth. The **periodontal ligament** surrounds the tooth root and extends from the apex of the root to the base of the **gingival sulcus**. The gingival sulcus, the space between the free gingiva and the tooth surface, is lined internally by a thin layer of epithelial cells. The base of the sulcus is formed by the **junctional epithelium**, a specialized type of epithelium that attaches the gingiva to the root. This epithelium provides the barrier to the ingress of bacteria. In health, the gingival sulcus is less than 3 mm deep as measured by a periodontal probe from the **cementoenamel junction (CEJ)** to the base of the sulcus. Colonization of bacteria within the sulcus promotes inflammation that eventually leads to breakdown of the **epithelial attachment**. Evidence of chronic inflammation is the apical extension of the epithelial attachment beyond 3 mm. Although accumulation of **bacterial plaque** is the most important factor influencing the health of the periodontium, position of the tooth within the arch, occlusal loading, parafunctional habits, appliances, drugs, and frenal attachments also affect periodontal health and the development of periodontal pockets.

Alveolar Mucosa and Frenal Attachments (Figs. 3.3 and 3.4) Mucosa is epithelium covering mucus-secreting glands. The **alveolar mucosa** is movable mucosa that overlies alveolar bone and borders the apical extent of the periodontium. It is movable because it is not bound down to the underlying periosteum and bone. The alveolar mucosa is thin and highly vascular. Accordingly, it appears pinkish-red, red, or bright red. On close inspection, small arteries and capillaries can be seen within the alveolar mucosa. These vessels provide nutrients, oxygen, and blood cells to the region. The mucosa is generally identified as either **buccal mucosa** (if it is located posteriorly) or **labial mucosa** (if it is located anteriorly).

Frena are lip and cheek muscle attachments at specific locations within the alveolar mucosa. They appear as arclike rims of flexible fibrous tissue when the lips or cheeks are distended. Six oral frena have been identified. The **maxillary labial frenum** is located at the midline between the maxillary central incisors, about 4

to 7 mm apical to the interdental region. The **mandibular labial frenum** appears similarly below and between mandibular central incisors within the alveolar mucosa. The two **maxillary** and two **mandibular buccal frena** are located within the alveolar mucosa near the first premolar on the right and left sides. Although frena do not directly contribute to periodontal support, those that attach within 3 mm of the CEJ of a tooth can pull on periodontal tissues and contribute to the development of gingival recession.

Mucogingival Junction (Fig. 3.5) The **mucogingival junction** is an anatomic landmark representing the border between the unattached alveolar mucosa and the attached gingiva. The junction is about 3 to 6 mm below the CEJ and extends around the buccal and lingual aspects of the arches. Visibility of the junction depends on the difference in vascularity and color of the two tissues. It is easily distinguished when the alveolar mucosa is red and the attached gingiva is pink.

Attached Gingiva and Free Marginal Gingiva (Figs. 3.6–3.8) The attached gingiva and free marginal gingiva cover the outer aspect of the gingival sulcus. The **attached gingiva** extends coronally from the alveolar mucosa to the free marginal gingiva. It is covered by keratinized epithelium, is bound down to periosteum, and cannot be moved. In health, the attached gingiva is pink and 2 to 7 mm wide. Its surface is slightly convex and stippled, like the surface of an orange. **Interdental grooves** can be seen in the attached gingiva as vertical grooves or narrow depressions located between the roots of the teeth.

The **marginal gingiva** provides the gingival collar around the cervix of the tooth. It is pink and keratinized like the attached gingiva, with a smooth rounded edge. Unlike the attached gingiva, the marginal gingiva is not attached to periosteum, nor is it stippled. Its freely movable nature allows a periodontal probe to be passed under it during assessment of pocket depth. Accordingly, it is also termed the **free marginal gingiva**. The junction between the marginal gingiva and the attached gingiva is called the **free gingival groove**.

The **interdental papilla** is the triangular projection of marginal gingiva that extends incisally between adjacent teeth. The papilla has a buccal and lingual surface and an interdental region (the **col**) that is concave, depressed, and covered by free marginal gingiva. In health, papillae are pink and knife-edged, can barely be moved by the periodontal probe, and extend near to the interdental contact region. The presence of inflammation and disease (i.e., gingivitis) alter the color, contour, and consistency of the free marginal gingiva and interdental papillae, causing the marginal gingiva to appear purple, soft, swollen, and tender, and the papillae to relax away from the tooth.



Fig. 3.1. Healthy periodontium: anterior view.



Fig. 3.2. Healthy periodontium: lingual aspect.



Fig. 3.3. Healthy periodontium and buccal frenum.



Fig. 3.4. Red alveolar mucosa and labial frenum.



Fig. 3.5. Mucogingival junction: identified by arrow.

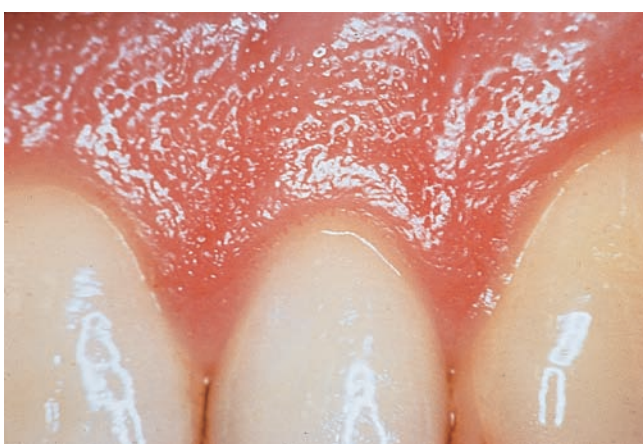


Fig. 3.6. Attached gingiva: stippled texture.



Fig. 3.7. Interdental grooves.

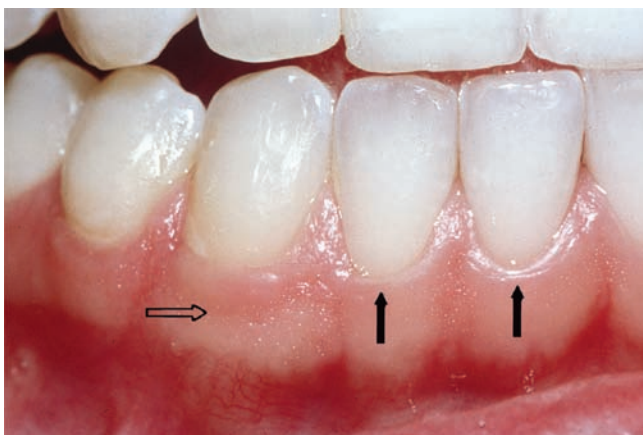


Fig. 3.8. Marginal gingiva and gingival groove, open arrow.

OCCUSION AND MALOCCLUSION

Occlusion is the relation of the maxillary and mandibular teeth during functional contact. The term is used to describe the way teeth are aligned and fit together. In an ideal occlusion, all the maxillary teeth fit slightly over the mandibular teeth, the cusps of the upper molars fit into the buccal grooves of the lower molars, and the midline is aligned. Few people have perfect occlusion, and **malocclusion** (abnormal positional relationship of the maxillary teeth with the mandibular teeth) is a common reason for patients to seek orthodontic care. Although most malocclusions do not require treatment, correcting a malocclusion can enhance the patient's appearance and ability to clean their teeth, and reduce the risk of developing oral disease.

Malocclusion is often hereditary. It results when the upper and lower jaws are disproportionate in size, the size of the teeth is too large or small for the jaws, or the spacing/eruption of teeth is abnormal. The following is a summary of the modified classification of occlusion first established by the orthodontist Edward Hartley Angle, who based his classification (**Angle's classification**) on the occlusal relationships of the permanent first molars.

Class I Occlusion (Figs. 4.1–4.3) Class I is considered to be the ideal (normal) occlusion and normal anteroposterior relationship of the jaws. In Class I occlusion, the mesiobuccal cusp of the permanent maxillary first molar occludes (fits) into the buccal groove of the permanent mandibular first molar. Also, the maxillary canine occludes into the interproximal space between the mandibular canine and first premolar.

Class II Occlusion (Figs. 4.4–4.9) Class II occlusion occurs when the maxillary teeth appear anterior to the normal relationship with the mandibular teeth. In Class II occlusion, the mesiobuccal cusp of the permanent maxillary first molar occludes mesial (anterior) to the buccal groove of the permanent mandibular first molar. Variants include **Class II Division 1**, in which the maxillary teeth are protruded (labioversion) and the maxillary first molar is anterior to the normal relationship, and **Class II Division 2**, in which the maxillary central incisors are intruded (linguoversion) and the maxillary first molar is anterior to the normal relationship.

Class III Occlusion (Figs. 4.10–4.12) These patients typically have a prognathic profile (the lower jaw projects forward). The mesiobuccal cusp of the permanent maxillary first molar occludes distal (posterior) to the buccal groove of the permanent mandibular first molar. A Class III malocclusion is seen in about 3 % of the U.S. population.

Overbite: The vertical overlap of the maxillary teeth over the mandibular teeth when the posterior teeth are in contact in centric occlusion

Overjet: The horizontal overlap (protrusion) of the maxillary anterior/posterior teeth beyond the mandibular teeth when the mandible is in centric occlusion

Subdivision: A unilateral condition on the left or right side only

Note: patients can have different classes of malocclusion on the left and right sides.



Fig. 4.1 Angle Class I: normal occlusion, right.



Fig. 4.4 Angle Class II division 1: malocclusion, right.



Fig. 4.7 Angle Class II division 2: malocclusion, right.



Fig. 4.10 Angle Class III: malocclusion, right.



Fig. 4.2. Angle Class I: normal occlusion, center.



Fig. 4.3. Angle Class I: normal occlusion, left.



Fig. 4.5. Angle Class II division 1: malocclusion, center.



Fig. 4.6. Angle Class II division 1: malocclusion, left.



Fig. 4.8. Angle Class II division 2: malocclusion, center.



Fig. 4.9. Angle Class II division 2: malocclusion, left.



Fig. 4.11. Angle Class III: malocclusion, center.



Fig. 4.12. Angle Class III: malocclusion, left.

RADIOGRAPHIC LANDMARKS: MAXILLA

Anterior Midline Region (Figs. 5.1 and 5.2) The anterior maxillary image contains several important anatomic landmarks and structures. The **incisive foramen** is an ovoid depression in the anterior midline of the **hard palate** that contains the nasopalatine nerve and blood vessels. Radiographically, it appears as an ovoid radiolucency with a fine radiopaque margin. The foramen overlies the median palatal suture and is located between the roots of the central incisors. The **median palatal suture** appears as a midline radiolucent line bordered by a radiopaque margin. It runs vertically and apically between the roots of the central incisors to the V-shaped **anterior nasal spine**. The **soft tissue outline of the nose** extends to the apices of the incisors, and the **soft tissue outline of the upper lip** is often seen as a light radiopacity bisecting the crowns of the central incisors. Alveolar bone in this region appears as fine, interspersed radiopaque **trabeculae** that surround radiolucent **marrow spaces**. The **cementoenamel junction (CEJ)**, or cervical line, of the incisors is seen as a smooth, round line delineating the crown and root portions of the tooth. Apically, the CEJ is a more subtle round line above the **crest of the alveolar bone**. In Figure 5.2, root structure between the CEJ and alveolar crest is not covered by bone owing to destruction by periodontal disease.

Anterior Lateral Region (Fig. 5.3) The **superior foramen of the incisive canal** is seen as a round radiolucent landmark within the **nasal fossa** and above the root apex of the central incisor and the radiopaque line representing the **floor of the nasal fossa**. The radiolucent **incisive canal** runs vertically below the incisive foramen. The **soft tissue outline of the nose** is seen bisecting the roots of the central and lateral incisors. The radiolucent **periodontal ligament space** and radiopaque **lamina dura** surround the roots. The crowns demonstrate a radiopaque **enamel** outer layer, a less dense inner layer of **dentin**, and a centrally located radiolucent **pulp chamber**. Each tooth root has an outer layer of **cementum** that is not normally visible on radiographs, unless excessive amounts, called **hypercementosis**, are present. Beneath the cementum is the dentin of the root that appears immediately adjacent to the radiolucent periodontal membrane space. Centrally within the root is the **root canal space(s)**. In the central and lateral incisors, note the **cervical line** crossing the junction between the crown and roots of the teeth. Because of the excess vertical angulation of the beam in this example, the buccal cervical line is projected downward and the lingual cervical line is projected upward. Distal to the lateral incisor is a slightly more radiolucent area called the **lateral fossa**, which is a depres-

sion on the labial bone between the lateral and canine roots.

Canine Region (Fig. 5.4) The **inverted Y** is prominently seen in the top portion of the canine image. It is composed of two structures: the floor of the nasal cavity (fossa) and the anterolateral wall of the maxillary sinus. The more anterior arm of the inverted Y consists of the floor of the nasal cavity (fossa); the more posterior curved arm is the anterolateral wall of the maxillary sinus. The **soft tissue outline of the nasal mucosa** is delineated by a thin radiolucent line representing an air space between the nasal turbinate and nasal mucosa.

Premolar Region (Fig. 5.5) The **floor of the maxillary sinus** is located above the premolar roots and in some cases the molar roots. The normal floor of the maxillary sinus appears as an irregular, slightly wavy line. Above the floor and within the lateral sinus wall is the curved radiolucent line representing the **canal of the posterior superior alveolar nerve, artery, and vein**. Notice that this canal has thin radiopaque margins. Above the second molar root is the radiopaque **zygomatic process of the maxilla**, sometimes referred to as the **malar process**. It is the anterior root of the zygomatic arch. Sometimes on a premolar image, the nasolabial fold bisects the root of the first premolar. Note the elongated palatal root of the first molar and the shortened buccal roots owing to incorrect positioning (excessive vertical angulation) of the **beam-indicating device (BID)** during image exposure.

Molar Region (Fig. 5.6) A prominent landmark in the maxillary molar image is the radiopaque U-shaped “malar shadow,” which is the zygomatic process of the maxilla. It delineates the most anterior extent of the **zygomatic arch** (cheekbone). The zygomatic arch is buccal and lateral to the maxilla and extends horizontally across the upper portion of the molar image. In this example, it extends across the posterior portion of the **maxillary sinus**. Distal to the second molar is the **maxillary tuberosity**—a bony structure covered by connective tissue and mucosa.

Tuberosity Region (Figs. 5.7 and 5.8) Distal to the second molar is the **maxillary tuberosity**, the **lateral pterygoid plate**, and small **hamular process** of the medial pterygoid plate. Superior and lateral to this region is the **zygomatic arch**. The anterior half of the zygomatic arch is delineated from the posterior portion by the **zygomaticotemporal suture** (Fig. 5.7). The **coronoid process** of the mandible is seen overlying the inferior portion of this region (Figs. 5.6–5.8).



Fig. 5.1. Maxilla: lingual aspect of central incisor region.



Fig. 5.2. Maxilla: central incisor region radiograph.



Fig. 5.3. Maxilla: lateral incisor radiograph.



Fig. 5.4. Maxilla: canine periapical image.



Fig. 5.5. Maxilla: premolar periapical image.



Fig. 5.6. Maxilla: molar periapical image.



Fig. 5.7. Maxilla: tuberosity region on skull.

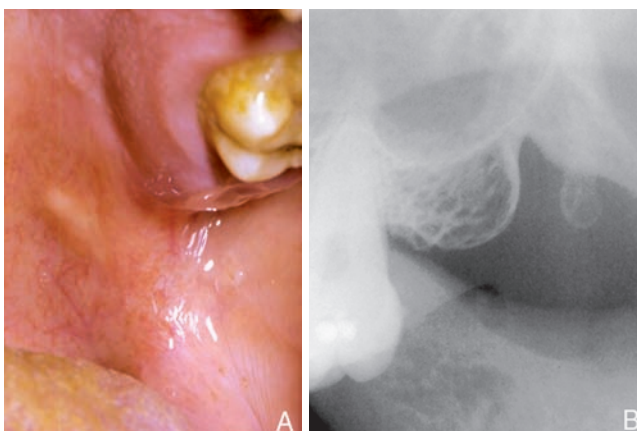


Fig. 5.8. Maxilla: Clinical photograph (A) and radiograph (B) of tuberosity region.

RADIOGRAPHIC LANDMARKS: MANDIBLE

Incisor–Canine Region (Figs. 6.1 and 6.2) On the lingual aspect of the mandible, the incisor image reveals the **lingual foramen** located several millimeters below the root apices. This radiolucent landmark is surrounded by the four **genial tubercles**. The superior tubercles serve as the attachment site of the **genioglossus muscle**, and the inferior pair anchors the **geniohyoid muscle**. The **inferior border** of the mandible below this area is delineated by a thick cortex (outer covering). Radiographically, the **genial tubercles** appear as round doughnut-shaped radiopacities. In this case, the **lingual canal** extends inferiorly from this region. Below this is the **inferior cortex** of the mandible. The inverted V-shaped thick radiopaque line along the incisor root apices is the **mental ridge**; it is located on the buccal aspect of the mandible.

Premolar and Molar Regions (Figs. 6.3 and 6.4) In the photographs of the skull, the **mental foramen** is located near the root apex of the second premolar, and the **external oblique ridge** is highlighted distal to the second molar. Both are landmarks of the buccal aspect of the mandible. On the lingual side of the mandible is the **internal oblique** or **mylohyoid ridge**. It is anterior, more horizontal, and longer than the external oblique ridge. Beneath the mylohyoid ridge is a **fossa** or **depression** within which lies the submandibular salivary gland.

Premolar Region (Fig. 6.5) Radiographically, the **mental foramen** is a round or ovoid radiolucency about 2 to 3 mm in diameter that lacks a distinct radiopaque corticated margin. Its location varies from the distal aspect of the canine to the distal aspect of the second premolar in the apical region. In this radiograph, a mixed **trabecular pattern** is seen with a denser (more radiopaque) pattern toward the alveolar crest and a looser (more radiolucent) pattern in the apical area. Loose and dense trabecular patterns depend on the number of bone trabeculae present in the region. In this radiograph, the radiopaque **lamina dura** and radiolucent **periodontal membrane space** are well illustrated in the second premolar. The radiopaque **crestal alveolar bone** between the premolars is healthy. When the alveolar bone starts to resorb as a result of periodontal disease, the crestal bone (radiopaque line) is lost. The densely radiopaque material in the crowns of the second premolar and molar is **amalgam**. Notice that the gingival margins of the restorations are smooth and continuous with the remaining tooth structure in the interproximal areas, which helps to maintain proper periodontal health. In this view, the buccal cusps are slightly higher than the lingual cusps owing to excess negative vertical angulation of the BID during the exposure.

Buccal Aspect Molar Region (Fig. 6.6) The **external and internal oblique ridges** are densely radiopaque structures that sometimes parallel each other. The external oblique ridge is above and posterior to the internal oblique ridge. The smooth, round radiopaque area at the bifurcation of the first molar is frequently mistaken for an enamel pearl or pulp stone. Actually, it is an anatomic artifact (due to superimposition of buccal and lingual root structure at the bifurcation) produced by incorrect horizontal angulation of the BID. The artifact disappears when the correct horizontal angulation of the BID is used—in cases in which it does not disappear, an enamel pearl or pulp stone should be suspected.

Lingual Aspect Molar Region (Fig. 6.7) The **submandibular fossa** is a broad radiolucent area immediately beneath the mylohyoid ridge and above the **inferior cortex**. It is seen more often when excessive negative vertical angulation of the BID is used.

Internal Aspect Molar Region (Fig. 6.8) The **inferior alveolar canal**—containing the inferior alveolar nerve and blood vessels—appears as a radiolucent canal in the molar image. The canal is outlined by parallel radiopaque cortical lines representing the canal walls and may be in close proximity to the molar apices or to developing third molars. This close relationship to third molars is important when considering the removal of the third molars. A **stepladder trabecular pattern** is sometimes seen between the roots of mandibular first molars (and central incisors). This usually represents a variation of normal. However, if generalized in appearance, it may indicate a severe form of anemia. In this instance, the trabeculae are horizontal, in a limited region, and more or less parallel to each other. Note the fractured distal surface of the first molar, the subtle occlusal caries in the second molar, and the developing third molar.

Author comment: We purposely used no. 2 size image in these examples to provide as many landmarks as possible in the limited space available. Some views in the standard full-mouth radiographic series were omitted because of space limitations. Similar landmarks can be seen in the narrower and popular no. 1 image. Also, some landmarks are seen variably, depending on individual patient differences and whether the bisecting angle or paralleling techniques are used or whether excessive vertical or horizontal angulation of the BID is used. Landmarks and structures are not indicated by arrows as they can obscure adjacent anatomic structures.

Remember, the recognition of normal is an absolute prerequisite to recognizing and identifying disorders and diseases. As we have often said, learning should be fun, and we hope this descriptive and illustrative approach helps.



Fig. 6.1. Mandible: lingual aspect incisor-canine region.



Fig. 6.2. Mandible: incisor-canine periapical image.



Fig. 6.3. Mandible: external oblique ridge; see Fig. 6.6.



Fig. 6.4. Mandible: internal oblique ridge; see Fig. 6.6.



Fig. 6.5. Mandible: premolar periapical image.

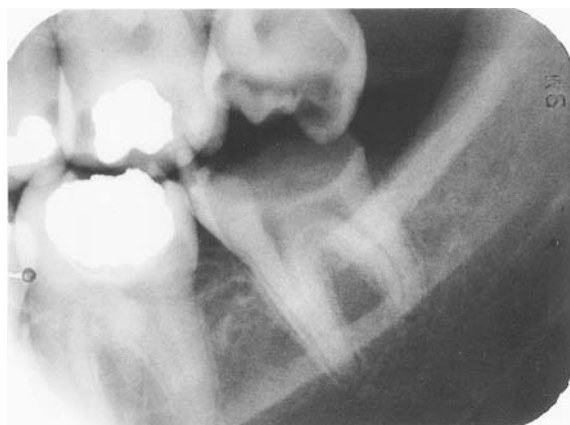


Fig. 6.6. Mandible: molar periapical image.



Fig. 6.7. Mandible: molar periapical image.



Fig. 6.8. Mandible: molar periapical image.

TEMPOROMANDIBULAR JOINT

Normal Anatomy (Figs. 7.1–7.2) The **temporomandibular joint (TMJ)** is composed of several major hard and soft tissue structures. The bony structures (visible in radiographic images) include the head of the **condyle** and condylar **neck**. The soft tissue components, shown in the diagram (Fig. 7.1) and anatomic specimen (Fig. 7.2), include the **disk** and **joint capsule**. The disk is made of fibrous cartilage disk, is hourglass shaped, and lies above the condyle and below the glenoid fossa. The disk is located within the joint capsule that contains the synovial fluid. The disk and synovial fluid cushion the head of the condyle from the bones of the glenoid fossa. The disk divides the joint capsule into the upper and lower joint spaces. It is attached posteriorly to the joint capsule, superiorly to the temporal bone, inferiorly to the posterior condyle, and anteriorly to the capsule and external pterygoid muscle. When the jaws are closed, the condyle is centered in the glenoid fossa of the temporal bone. During opening, the condyle first “rotates” in the glenoid fossa then “translates” as the mouth opens wider. Upon normal maximum opening, the condylar head approximates the articular eminence of the base of the skull.

All of the components of the TMJ are subject to functional and/or pathologic change. Some of the major clinically observable features of TMJ function or dysfunction are illustrated. The major observable **signs** of TMJ disorders are swelling in the TMJ area; redness of the overlying skin; pain/tenderness to palpation of the TMJ; atrophy, hypertrophy, or paralysis of the muscles of mastication; pain on palpation of the muscles of mastication or their attachments; abnormal audible sounds, such as popping or crepitus (grinding); facial asymmetry; occlusal abnormalities, such as unilateral posterior open bite (apertognathia); crossbite; acquired anterior open bite; a shift in the anterior midline; and radiographic changes. Common **symptoms** elicited with TMJ disorders include reports of popping (or crepitus) sounds; pain at rest, on opening, or on chewing; limited opening; ringing in the ear; headaches or earaches; changes in the face, such as “my face or jaw looks crooked or swollen”; inability to chew or eat properly; and the inability to fully close the jaw.

Normal Opening (Fig. 7.3) **Normal maximum opening** is expressed in terms of how much and the amount of deviation. The “how much” is usually expressed in millimeters (mm) measured between the incisal edges of the upper and lower central incisors. Normal opening in a healthy adult is usually at least 40 mm. However, patients vary greatly in size, and a simple quick assessment can be made by asking the patient if he or she can

open fully to accommodate three fingers (the index, middle, and ring fingers) between the incisal edges of the maxillary and mandibular teeth. Limited opening consists of a width less than three fingers, but seldom are functional reports made unless the opening is severely restricted (<2 fingers).

Deviation on Opening (Fig 7.4) The assessment of **deviation** is performed by observing the relationship of the mandibular midline (between the central incisors) with the maxillary midline during opening. When the midlines do not line up during opening, this is called deviation. Deviation can occur to one side only, or first to one side, then the other.

Posterior Open Bite–Midline Deviation–Crossbite (Fig. 7.5) **Posterior open bite** is also referred to as apertognathia. The term *ipsilateral apertognathia* is used when the open bite is on the same side as the TMJ disorder (usually a tumor). The term *contralateral apertognathia* is used when the open bite is on the opposite side as the TMJ problem. This may happen after condylectomy or TMJ fractures. In Figure 7.5, the patient is in centric occlusion; he had an ipsilateral apertognathia, deviation of the midline at rest, and a crossbite that is due to an osteochondroma on his right condyle.

Anterior Open Bite (Fig. 7.6) Patients can have an **anterior open bite** from childhood habits such as tongue thrusting or thumb sucking. In these instances, the mamelons of the incisors may persist well into adult life. Anterior open bite is also seen with certain developmental anomalies of the TMJ and conditions that damage the TMJ condyle or condylar neck. Bilateral fractures of the condyles or bilateral condylectomies are traumatic causes of anterior open bite. One of the most common causes of anterior open bite in aging adults is resorption of the condyles because of degenerative diseases such as rheumatoid arthritis. With this disease, the superior condylar surface is slowly destroyed, producing wear facets and a loss of vertical height of the head of the condyles.

Crossbite (Figs. 7.7 and 7.8) A **crossbite** can be a sign of a TMJ abnormality or neoplasm. In Figure 45.3 there is a crossbite that is due to unilateral enlargement of the tongue (hemihypertrophy). Some patient’s hemihypertrophy involves the condylar neck, making this structure longer on one side than the other. In Figures 7.7 and 7.8, a growth deficit resulted in the contralateral crossbite readily observed, which contributed to facial asymmetry, especially evident in the lower third molar region.

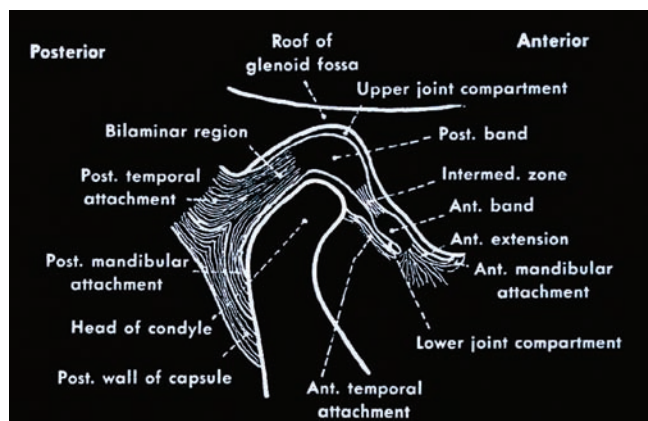


Fig. 7.1. TMJ: anatomy diagram of hard and soft tissues.



Fig. 7.2. TMJ: anatomic section; correlate with Fig. 7.1.



Fig. 7.3. TMJ: normal opening; patient's three fingers.



Fig. 7.4. TMJ: limited opening with significant deviation.



Fig. 7.5. TMJ: posterior open bite, midline deviation, and crossbite.



Fig. 7.6. TMJ: anterior open bite in rheumatoid arthritis.



Fig. 7.7. TMJ: developmental crossbite; long condylar neck.

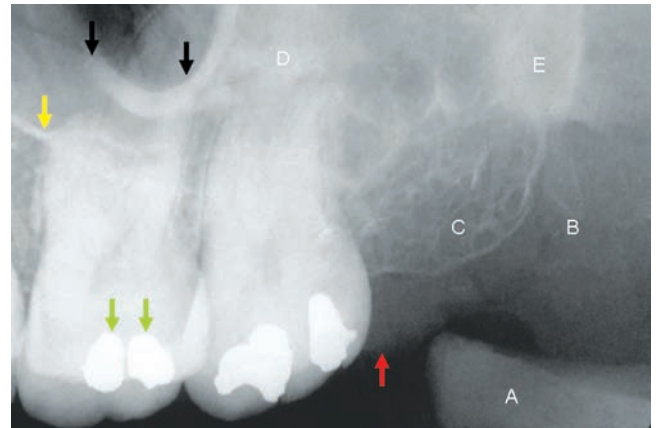


Fig. 7.8. TMJ: facial asymmetry in same patient as Fig. 7.7.

CASE STUDIES

CASE 1. (Fig. 7.9)

1. Identify what region is shown in this periapical image.
2. Identify the structure labeled *A*.
3. Is this a normal finding of the maxilla?
4. Identify the structures labeled *B* and *E*.
5. Identify the structure labeled *C*.
6. Identify the structure labeled *D*.
7. Identify the structure labeled by the black arrows.
8. Identify the structure labeled by the yellow arrow.
9. Identify the structures labeled by the green and red arrows.



CASE 2. (Fig. 7.10) This 22-year-old young lady presents to your dental office for routine dental care.

1. Identify the structure labeled *A*.
2. Identify the structure labeled by the yellow arrows.
3. Identify the structure labeled by the green arrow. Is this structure normal or abnormal, and how is it contributing to the dentition?
4. Identify the gingival tissue that is light brown in color. Why is it brown? Is this a normal finding, a variant of normal, or disease?
5. True or false: The marginal gingiva in this patient is pigmented.
6. Identify the structure labeled by the black arrow.
7. Identify the structure labeled by the red arrow. Is this structure healthy or diseased?



Diagnostic and Descriptive Terminology

Dental Objectives:

- Recognize, define, and use diagnostic terms that describe:
 - a. Colored or textured lesions that can occur in the skin and oral mucosa
 - b. Lesions that denude epithelium
 - c. Lesions associated with inflammatory reactions
 - d. Clefts and cavities within skin and oral mucosa
 - e. Solid raised lesions in the skin and oral mucosa.
 - f. Fluid-filled lesions in the skin and oral mucosa
- Recognize clinical features of skin and mucosa associated with infection and inflammatory disease.
- Recognize clinical features of benign and malignant disease.

Dental Hygiene Objectives:

- Define the descriptive terminology presented in this section.
- Use the descriptive terminology from this section to accurately describe and document extraoral and intraoral findings in a clinical setting.

DIAGNOSTIC AND DESCRIPTIVE TERMINOLOGY

Macule (Figs. 8.1 and 8.2) A **macule** is a small confined area of epidermis or mucosa distinguished by color from its surroundings. By definition, it is <1 cm in diameter. The macule may appear alone or in groups as a red, blue, brown, or black stain or spot. It is neither elevated nor depressed. A macule may represent a normal condition, a variant of normal, or local or systemic disease. The term *macule* would be used to clinically describe the following conditions: oral melanotic macule, ephelis, amalgam, india ink or pencil tattoos, and focal argyrosis. The color and shape of the macule aids in the diagnosis. Conditions that appear as macules are discussed in detail under “Pigmented Lesions” (Figs. 66.1–68.8).

Patch (Figs. 8.3 and 8.4) A **patch** is a circumscribed area that is larger than the macule and differentiated from the surrounding epidermis by color, texture, or both. Like the macule, the patch is neither elevated nor depressed. Focal argyrosis, lichen planus, mucous patch of secondary syphilis, and snuff dipper’s patch represent patchlike lesions that may be seen intraorally. Conditions that appear as patches are discussed in detail under “White Lesions” (Figs. 57.1–59.8), “Pigmented Lesions” (Figs. 66.1–68.8), and “Sexually Transmissible Conditions” (Figs. 80.1–82.8).

Erosion (Figs. 8.5 and 8.6) **Erosion** is a clinical term that describes a soft tissue lesion in which the skin or mucosa is denuded (i.e., the epithelium is worn away

or destroyed). Erosions are moist and slightly depressed and often result from a broken vesicle, epithelial breakdown, or trauma. In the eroded area, the epithelium above the basal cell layer (layer above the connective tissue or dermis) is lost. Healing rarely results in scarring because the basal layer of epithelium remains intact. Pemphigus, erosive lichen planus (desquamative gingivitis), and erythema multiforme are diseases that produce mucocutaneous erosions. Conditions that appear as erosions are discussed in detail under “Vesiculobullous Lesions” (Figs. 72.1–76.8).

Ulcer (Figs. 8.7 and 8.8) An **ulcer** is a craterlike lesion of the skin or oral mucosa. It is the term used to describe an uncovered wound of cutaneous or mucosal tissue that exhibits gradual tissue disintegration and necrosis. The border of a mucosal ulcer is often round but can be irregular. Ulcers extend deeper than erosions, from beyond the basal layer of the epithelium into the dermis (connective tissue). Scarring may follow healing of an ulcer. Ulcers may result from trauma; aphthous stomatitis; infection by viruses, such as herpes simplex, variola (smallpox), and varicella-zoster (chickenpox and shingles); cancer; or granulomatous disease. Ulcers are usually painful and often require topical or systemic drug therapy for effective management. Conditions that appear as ulcers are discussed in detail under “Vesiculobullous Lesions” (Figs. 72.1–76.8) and “Ulcerative Lesions” (Figs. 77.1–79.8).

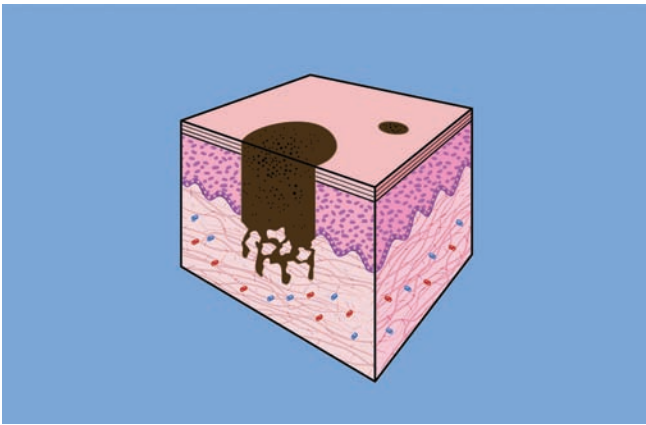


Fig. 8.1. Macule: nonraised area altered in color.



Fig. 8.2. Oral melanotic macule on the lip.

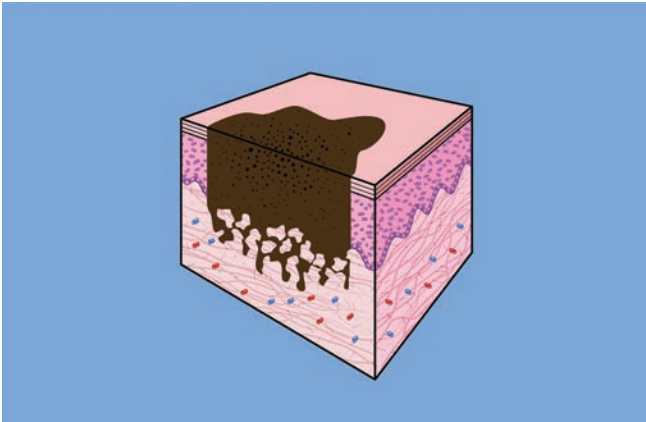


Fig. 8.3. Patch: pigmented area larger than a macule.



Fig. 8.4. Patch: amalgam tattoo after retrograde amalgam.

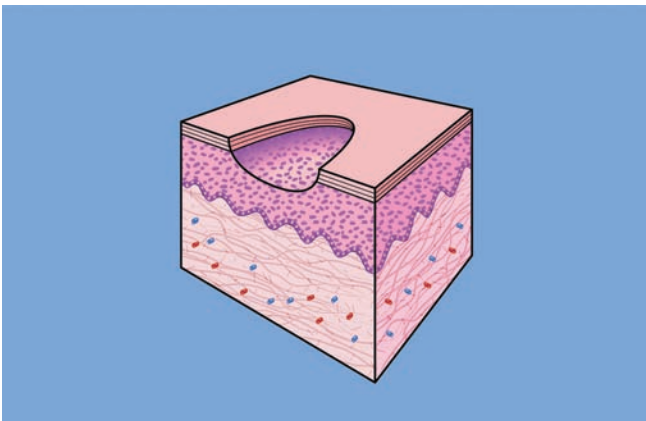


Fig. 8.5. Erosion: denudation above basal layer of epithelium.



Fig. 8.6. Erosion: erosive lichen planus on palatal gingiva.

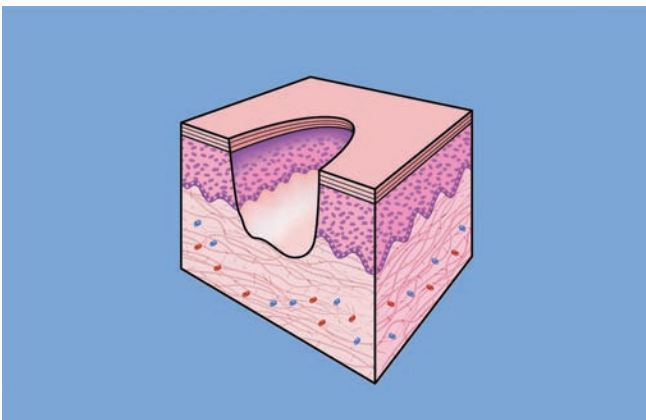


Fig. 8.7. Ulcer: denudation below basal layer of epithelium.



Fig. 8.8. Traumatic ulcer on lateral tongue border.

DIAGNOSTIC AND DESCRIPTIVE TERMINOLOGY

Wheal (Figs. 9.1 and 9.2) A **wheal** is a raised area of localized tissue swelling (edema). This smooth-surfaced papule or plaque results from acute extravasation of serum into the superficial dermis. Wheals are generally pale red, pruritic, and of short duration. By definition, they are only slightly raised and vary in size. They occur most commonly in persons with allergies. The wheal develops as a result of histamine release from mast cells or activation of the complement cascade. Wheals are a sign of an allergic reaction that develops shortly after insect bites, consuming a particular food, or mechanical irritation (such as that occurring in patients with dermatographia). They are often very itchy. Conditions that appear as wheals are discussed in detail under “Allergic Reactions” and “Vesiculobullous Lesions” (Figs. 74.1–78.8).

Scar (Figs. 9.3 and 9.4) A **scar** is a permanent mark or cicatrix remaining after a wound heals. These lesions are visible signs of wound repair and indicate a previous disruption in the integrity of the epidermis and dermis and healing of epithelium with fibrous (collagen connective) tissue. Scars are infrequently found in the oral cavity because oral tissue is elastic and less prone to scar formation than skin. When they do occur, they may be of any shape or size. They are not identical to the adjacent tissue. The color of an intraoral scar is usually lighter than that of the adjacent mucosa. Histologically, they are more dense than the adjacent epithelium, lack sweat (or salivary) glands, and have fewer blood vessels. Oral (i.e., periapical) surgery, burns, or intraoral

trauma may result in a scar. Scars are discussed in detail under “White Lesions” (Figs. 58.3–58.6).

Fissure (Figs. 9.5 and 9.6) A **fissure** is a normal or abnormal linear cleft or furrow in the epidermis (skin or mucosa) that affects the tongue, lips, and perioral tissues. The presence of a fissure can indicate a condition representing a variant of normal or disease. Disease-associated fissures result when pathogenic organisms infect a fissure, causing pain, ulceration, and inflammation. Fissured tongue is an example of a variation of normal that is associated with dry mouth and dehydration. Angular cheilitis and exfoliative cheilitis are examples of fissures associated with disease, specifically infection with *Candida albicans*.

Sinus (Figs. 9.7 and 9.8) The term **sinus** has two meanings. A common meaning of sinus is a normal recess or cavity, such as the frontal or maxillary sinus. The term is also used to describe as an abnormal dilated tract, channel, or fistula that leads from a suppurative cavity, cyst, or abscess to the surface of the epidermis. An abscessed tooth often produces a sinus tract that travels from the infected root apex to the clinically evident parulis, which is the terminal end of the tract. In this clinical situation, gutta percha points can be deeply placed into the tract and a radiograph taken. The nonvital tooth is identified by locating the tip of the gutta percha point adjacent to the nonvital root apex. Actinomycosis is a condition characterized clinically by several yellow sinus tracts exiting onto the mucosa or skin surface.

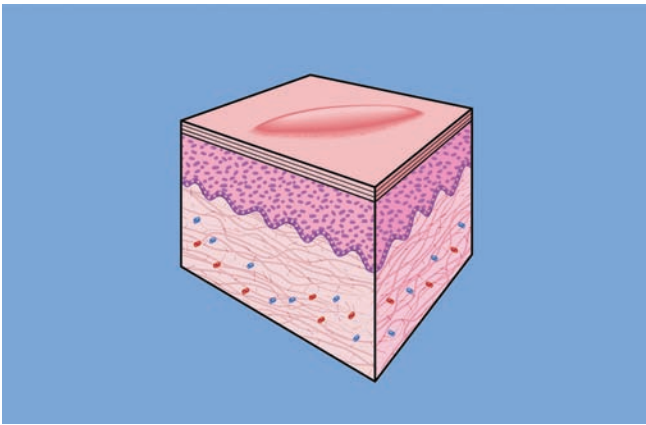


Fig. 9.1. Wheal: serum-filled papule or plaque.



Fig. 9.2. Wheal: dermatographism after rubbing the skin.

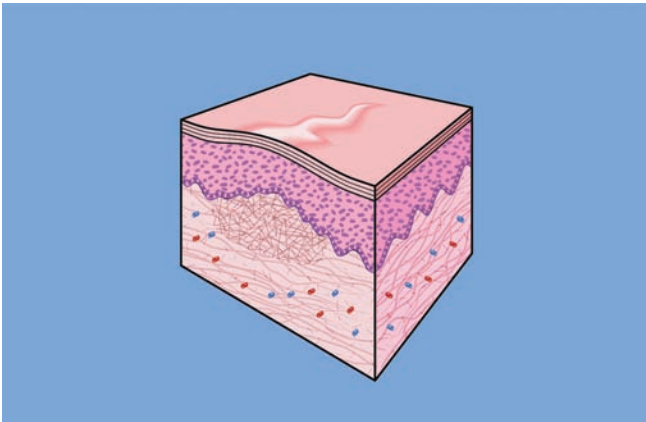


Fig. 9.3. Scar: permanent mark from wound.



Fig. 9.4. Scar: fibrotic tissue as a result of trauma.

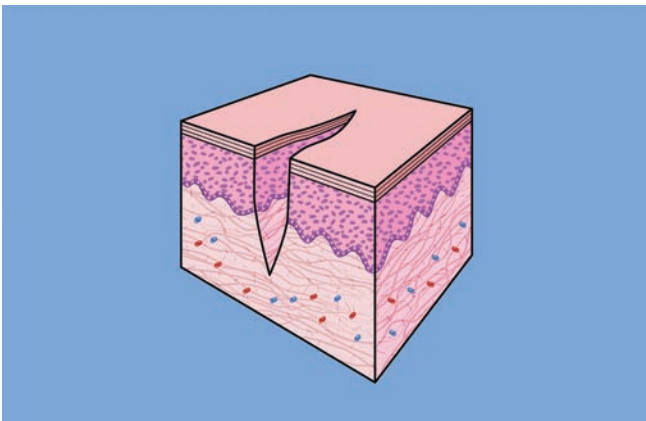


Fig. 9.5. Fissure: a linear crack in the epidermis.



Fig. 9.6. Fissure: fissured tongue, a variant of normal.

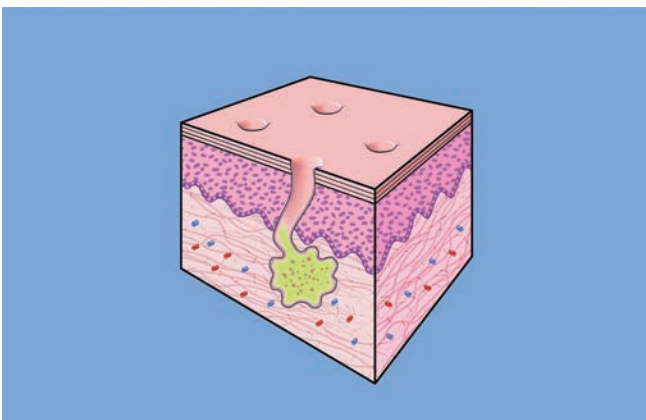


Fig. 9.7. Sinus: a recess, cavity, or dilated tract.



Fig. 9.8. Sinus tracts exiting from nonvital incisors.

DIAGNOSTIC AND DESCRIPTIVE TERMINOLOGY

Papule (Figs. 10.1 and 10.2) A **papule** is a small, superficial, elevated, solid lesion or structure that is <1 cm in diameter. Papules may be of any color and may be attached by a stalk or firm base. They often represent a benign or slow-growing lesion that is caused by infection, inflammation, hyperplasia, or neoplasia. Common examples of benign lesions appearing as papules include condyloma acuminatum, parulis, and squamous papilloma. Basal cell carcinoma, a slow-growing skin cancer, can also appear as a papule. Conditions that appear as papules are discussed in the section on “Papulonodules” (Figs. 71.1–71.8).

Plaque (Figs. 10.3 and 10.4) A **plaque** is a flat, solid, raised area of the skin or mucosa that is >1 cm in diameter. Although essentially superficial, plaques may extend deeper into the dermis than papules. The edges may be sloped, and sometimes keratin proliferates along the surface (a condition known as lichenification). Lichen planus, leukoplakia, or melanoma may initially appear as a plaque. Lichen planus is discussed under “Red and Red-White Lesions” (Figs 63.1–63.8). As with many medical-dental words, *plaque* has more than one definition. In dentistry, dental plaque is the biofilm of bacteria that builds up on teeth.

Nodule (Figs. 10.5 and 10.6) A **nodule** is a solid and raised lump or mass of tissue that has the dimension of depth. Like papules, these lesions are <1 cm in diameter; however, nodules extend deeper into the dermis. The nodule can be detected by palpation. The overlying epidermis is usually nonfixed and can be easily moved over the lesion. Nodules can be asymptomatic or painful and usually are slow growing. Benign mes-

enchymal tumors, such as fibroma, lipoma, lipofibroma, and neuroma, often appear as oral nodules. Other examples of nodules are discussed under “Nodules” (Figs. 69.1–70.8).

Tumor (Figs. 10.7 and 10.8) **Tumor** is a term used to indicate a solid mass of tissue >1 cm in diameter that has the dimension of depth. The term is also used to represent a **neoplasm**—a new, independent growth of tissue with uncontrolled and progressive multiplication of cells that have no physiologic use. Tumors may be any color and may be located in any intraoral or extraoral soft or hard tissue. Tumors are classified as **benign** or **malignant**. Benign tumors grow more slowly and are less aggressive than malignant tumors. Benign tumors often appear as raised, rounded lesions that have well-defined margins (clinically and radiographically); they do not metastasize. Malignant tumors are comprised of numerous neoplastic cells that have enlarged dark-staining (hyperchromatic) nuclei. The malignant tumor cells invade adjacent tissues and spread rapidly. Clinically and radiographically, they often have ill-defined margins. Persistent tumors may be umbilicated or ulcerated in the center. The term *tumor* is often used to describe a benign tissue mass such as a neurofibroma, granular cell tumor, or pregnancy tumor. The term **carcinoma** is reserved for malignant cancers of epithelial tissue (Figs. 79.3 and 79.4). The term **sarcoma** is reserved for a malignant neoplasm of embryonic connective tissue origin, such as osteosarcoma, a malignant neoplasm of bone. Malignancies destroy tissue by direct invasion and extension and by spread to distant sites by metastasis through blood, lymph, or serosal surfaces.

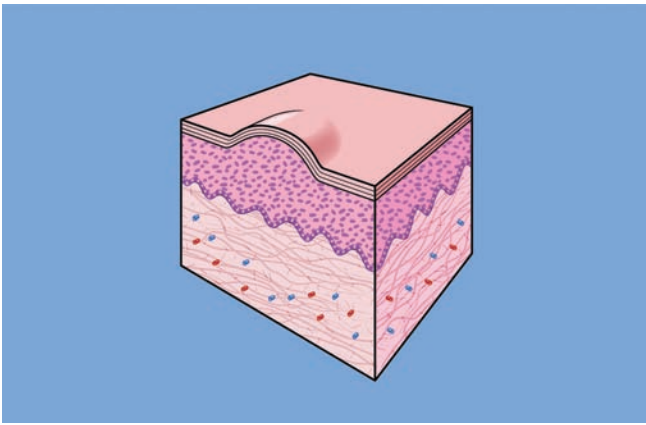


Fig. 10.1. Papule: elevated, solid lesion <1 cm wide.



Fig. 10.2. Papule: fibroepithelial polyp from chronic irritation.

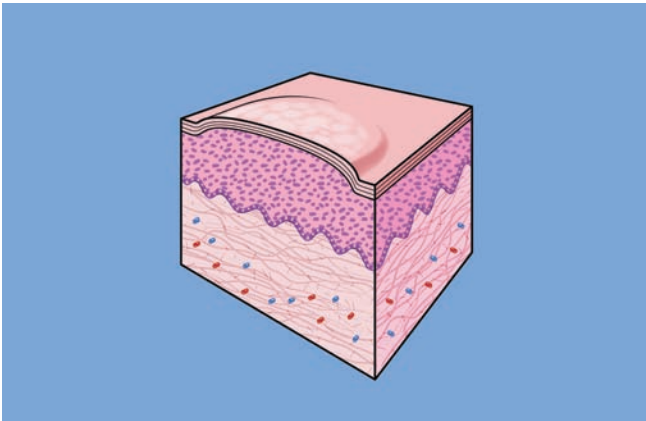


Fig. 10.3. Plaque: flat, raised area >1 cm in diameter.

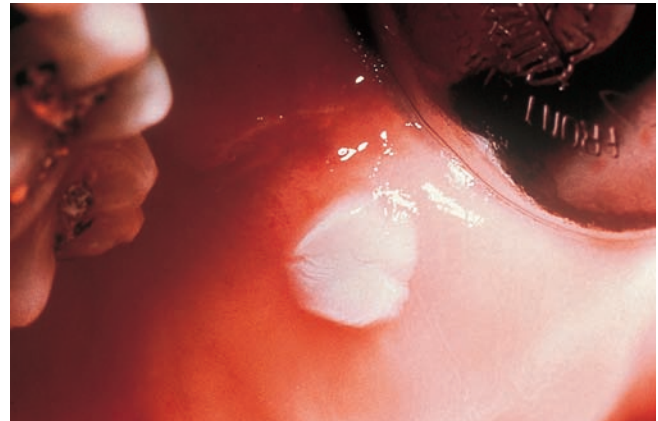


Fig. 10.4. Plaque: leukoplakia owing to clasp of appliance.

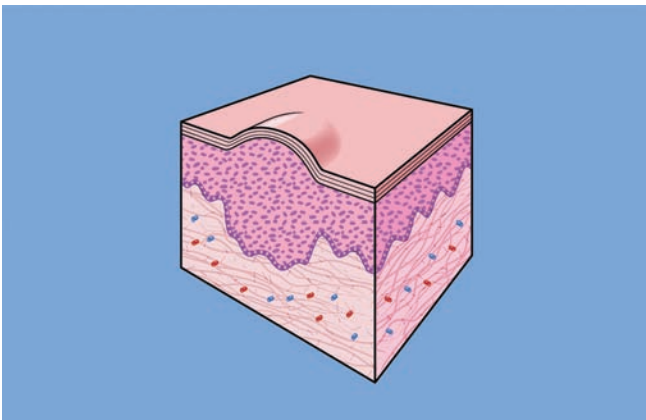


Fig. 10.5. Nodule: deep and raised mass <1 cm wide.



Fig. 10.6. Nodule: irritation fibroma at commissure.

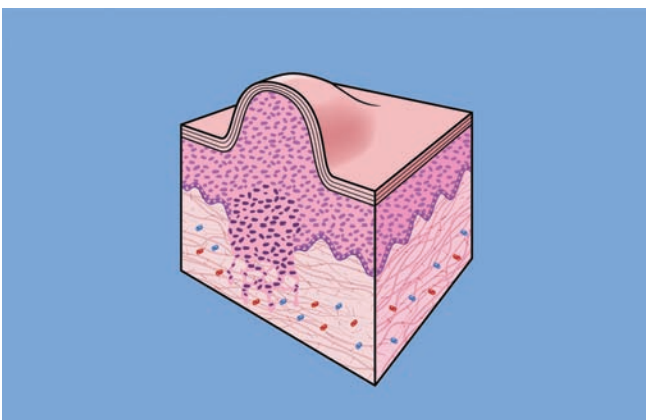


Fig. 10.7. Tumor: deep and solid mass >1 cm wide.



Fig. 10.8. Tumor: squamous cell carcinoma of tongue.

DIAGNOSTIC AND DESCRIPTIVE TERMINOLOGY

Vesicle (Figs. 11.1 and 11.2) A **vesicle** is a small, fluid-filled elevation in the epidermis (skin or mucosa) that is <1 cm in diameter. The fluid of a vesicle generally consists of lymph or serum but may contain blood and infectious agents. The epithelial lining of a vesicle is thin and will eventually break down, thus causing an ulcer and eschar (surface slough). Vesicles are a common result of inflammation caused by viral infections, such as herpes simplex, herpes zoster, chickenpox, and smallpox. In viral infections, the vesicle is laden with virus and is highly infectious. Conditions characterized by vesicles are discussed under “Vesiculobullous Lesions” (Figs. 72.1–72.8).

Pustule (Figs. 11.3 and 11.4) A **pustule** is a circumscribed elevation filled with pus—a purulent exudate consisting of a mixture of inflammatory cells and liquid—resulting from an infection. Pustules are <1 centimeter in diameter and may be preceded by a vesicle or papule. They are creamy white or yellowish and are often associated with an epidermal pore (i.e., pimple) or sweat gland. Within the mouth, a pustule is represented by a pointing abscess or parulis. Herpes zoster also produces pustules that eventually ulcerate and cause intense pain. See the discussions under “Localized Gingival Lesions” (Figs. 41.1 and 41.2), “Caries Progression” (Fig. 30.8), and “Vesiculobullous Lesions” (Figs. 73.1–73.4) for examples of diseases that produce pustules.

Bulla (Figs. 11.5 and 11.6) A **bulla** is a fluid-filled blister >1 cm in size. This condition develops from the accumulation

of fluid in the epidermal-dermis junction or a split in the epidermis. Accordingly, the surface is smooth and dome shaped and easily ruptured by the slightest of trauma. Because of their size, bullae represent a more severe disease than do conditions associated with vesicles. Bullae are commonly seen in pemphigus, pemphigoid, burns, frictional trauma, Stevens-Johnson syndrome, and epidermolysis bullosa. These conditions are discussed under “Vesiculobullous Lesions” (Figs. 76.1–76.8).

Cyst (Figs. 11.7 and 11.8) A **cyst** is a closed sac lined by epithelium (known as the capsule) located in the dermis, subcutaneous tissue, or bone. Cysts result from entrapment of epithelium or remnants of epithelium that grow to produce a cavity (the cavity portion is known as the lumen). Cysts range in diameter from a few millimeters to several centimeters. Aspiration of a cyst may or may not yield luminal fluid, depending on the nature of the cyst. Cysts that contain clear fluid appear pink to blue, whereas keratin-filled cysts often appear yellow or creamy white. Some of the many types of oral cysts are dermoid cysts, eruption cysts, implantation cysts, incisive canal cysts, lymphoepithelial cysts, mucus retention cysts, nasoalveolar cysts, radicular cysts, odontogenic keratocyst, dentigerous cyst, and the lateral periodontal cyst. Figure 11.8 demonstrates a gingival cyst, which is a peripheral variant of the lateral periodontal cyst. Bone cysts are discussed under “Radiolucent Lesions of the Jaws: Cysts” and “Tumors” (Figs. 31.1–31.8).

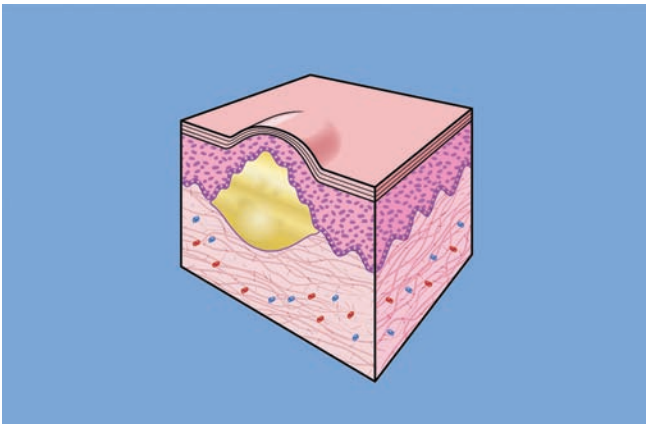


Fig. 11.1. Vesicle: small fluid-filled skin elevation.



Fig. 11.2. Vesicle: recurrent herpes simplex.

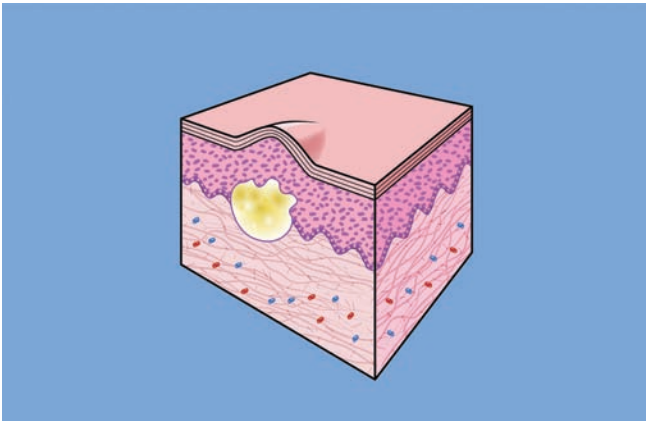


Fig. 11.3. Pustule: vesicle filled with purulent exudate.



Fig. 11.4. Pustule: pointing periodontal abscess.

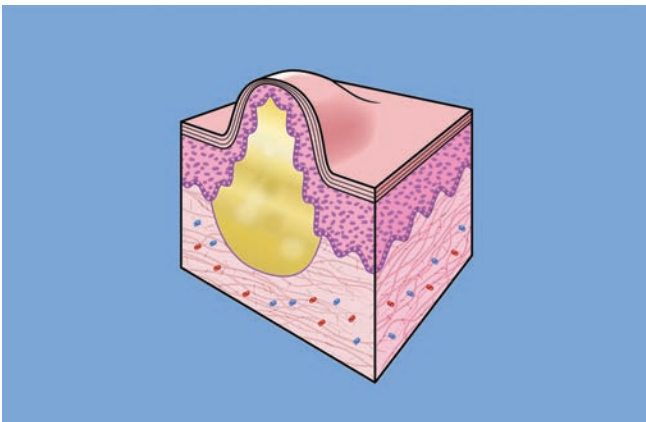


Fig. 11.5. Bulla: large fluid-filled mucocutaneous elevation.



Fig. 11.6. Bulla: bullous lichen planus—a rare finding.

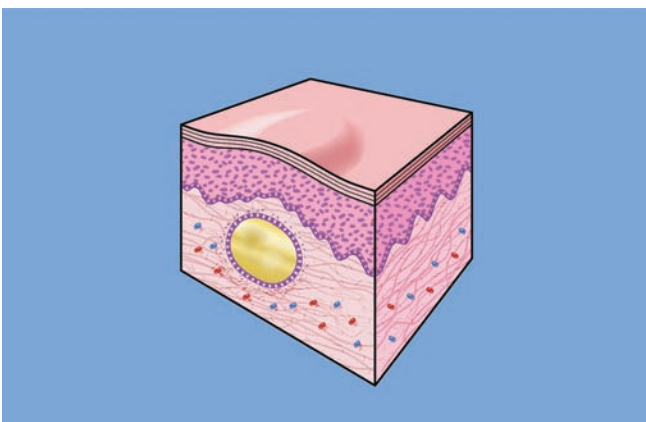


Fig. 11.7. Cyst: epithelial-lined cavity.



Fig. 11.8. Gingival cyst: variant of lateral periodontal cyst.

DIAGNOSTIC AND DESCRIPTIVE TERMINOLOGY

Normal (Figs. 12.1 and 12.2) **Normal** implies a tissue or part lacks significant deviation from the average. In other words, normal agrees with the regular established characteristics of appearance and function. Appearance includes color, shape, size, topography or architecture, consistency and temperature; histologically, it also includes characteristics seen on stained tissue specimens. Normal function implies the cells, tissue, or organs are performing as they should. For example, the normal oral mucosa is of a light pinkish color, is usually moist because of the presence of saliva, and can withstand the various normal functions of the mouth without injury or other abnormal change.

Hypotrophy and Atrophy (Figs. 12.3 and 12.4) **Hypotrophy** is the progressive degeneration of an organ or tissue caused by *loss in cell size*. The condition results when there is incomplete growth or maintenance of nutrition to a tissue or organ. Hypotrophy causes a diminution in the size of a tissue, organ, or part. **Atrophy** is similar in meaning to hypotrophy, but is often reserved for the eventual result of persistent conditions that led to hypotrophy. Atrophy is also referred to as a “wasting away” or a decrease in size of a body organ or tissue that is due to disease, lack of nourishment, injury, or lack of use. Examples of atrophy follow. The oral mucosa may become thinned when atrophic candidiasis develops. Muscles, such as the tongue or the muscles of mastication (masseter, temporalis, internal and external pterygoid muscles), may become atrophic as a result of disuse (disuse atrophy); muscle disuse may follow a loss of motor innervation that is due to trauma or following a stroke. Following the extraction of one, several, or all of the teeth, the alveolar bone of the mandible and/or maxilla will usually atrophy (diminish in size), providing less support for prostheses or sufficient bone for an implant. Continued atrophy of the alveolar bone results in impingement on vital structures, such as the mental nerve or the maxillary sinus.

Hypertrophy (Figs. 12.5 and 12.6) **Hypertrophy** implies the enlargement or overgrowth of a cell, tissue, organ, or part because of an *increase in size of its constituents or cells* without cell division. Hypertrophy may be a reactive process that is due to increased function or is genetically induced. Muscle hypertrophy is typically seen in body building, but can also occur in the muscles of mastication in persons who chronically clench or grind their teeth (bruxism). It is also seen in the mandibular condyle, which may increase in size as a result of hypertrophic arthritic or developmental changes. Reactive hypertrophy is seen in the contralateral salivary gland following the removal of the opposite salivary gland. Predetermined genetic change can be seen in bilateral hypertrophy of the coronoid process occurring in susceptible patients. Another example is seen in hemifacial hypertrophy as described in this text.

Hypoplasia (Figs. 12.7 and 12.8) **Hypoplasia** refers to an underdeveloped tissue or organ that has *decreased in number of cells or the amount of substance they produce or secrete*. The term is used when there is underdevelopment of an organ or tissue such that it fails to reach its full adult size. One dental example is enamel hypoplasia. This condition is associated with insufficient amount of enamel or the calcification within the enamel. Amelogenesis imperfecta is a condition associated with several hypoplastic variations that cause the presence of too little enamel, which may be smooth or pitted. Another example is focal dermal hypoplasia (Goltz-Gorlin syndrome). In this syndrome, the skin of the face appears depressed in places because of hypoplasia of the dermis, which is the tissue layer immediately beneath the skin. Condylar hypoplasia is a third example. In this case, the condyle of the temporomandibular joint is small and often deformed as a result of trauma or congenital defects affecting the cartilaginous growth center.

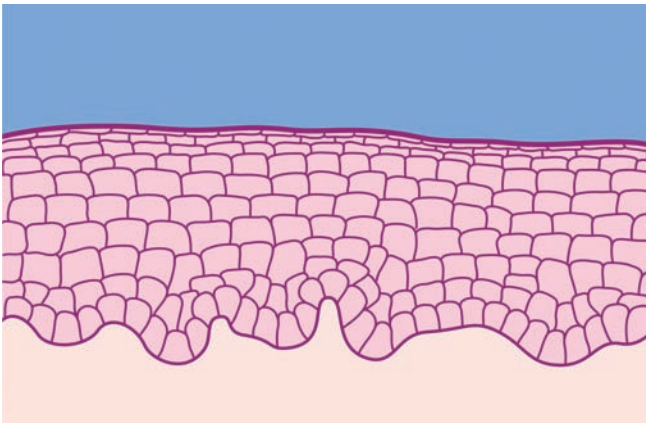


Fig. 12.1. Normal: appearance, number, size, architecture.



Fig. 12.2. Normal: soft tissues, gingiva, teeth, occlusion.

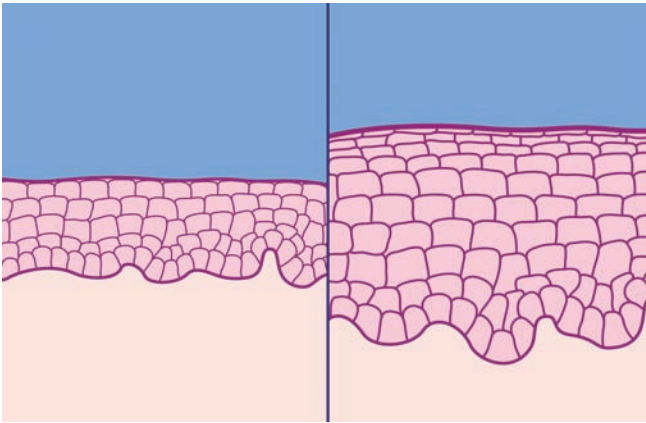


Fig. 12.3. Atrophy: decreased size of cells (left), normal (right).



Fig. 12.4. Atrophy: poststroke; tongue muscles atrophied.

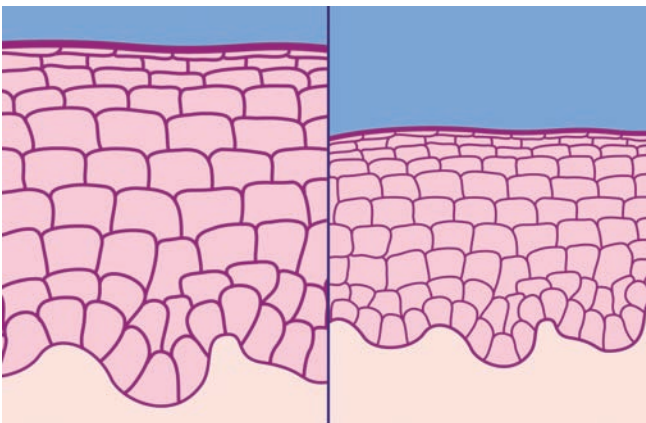


Fig. 12.5. Hypertrophy: increased size of cells (left), normal (right).



Fig. 12.6. Hypertrophy: tongue in facial hemihypertrophy.

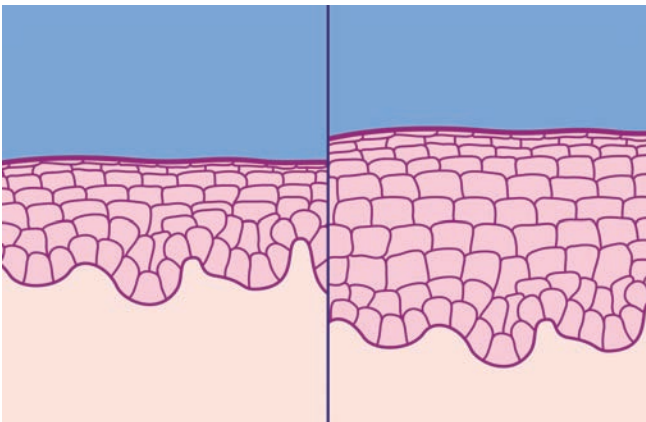


Fig. 12.7. Hypoplasia: decreased number of cells (left), normal (right).



Fig. 12.8. Hypoplasia: amelogenesis imperfecta; hypoplastic enamel.

DIAGNOSTIC AND DESCRIPTIVE TERMINOLOGY

Hyperplasia (Figs. 13.1 and 13.2) **Hyperplasia** is an *increase in the number of normal cells* in a tissue or organ that results in increased volume or size of the tissue or organ. The term implies there is an abnormal multiplication of cells that are normal in appearance, arrangement, and architecture. Hyperplasia is usually a reactive process secondary to some stimulus or growth factors. Puberty is a physiological stimulus that increases the size of the breast and gonadal tissue. In the oral cavity, inflammatory papillary hyperplasia is a reactive tissue growth that develops beneath a denture. Clinically, one sees numerous papillary growths on the palate that represent squamous epithelium increased in size. Chronic hyperplastic pulpitis (a pulp polyp, Fig. 30.2) is another example of this condition.

Metaplasia (Figs. 13.3 and 13.4) **Metaplasia** is the *replacement of one adult cell type with that of another adult cell type* not normal for that tissue. It often occurs as a form of adaptation to a stressful environment. One of the more common examples is the metaplastic change of respiratory columnar epithelium to squamous epithelium in response to chronic irritation (cigarette smoking). In another example, necrotizing sialometaplasia, there is squamous (epithelial) metaplasia of the cells lining the ducts of accessory salivary glands usually in the hard or soft palate. That is to say, the normal central lumen of the duct disappears and is replaced with cells that now look like small clusters of epithelium within the surrounding connective tissue. Another type of metaplasia is seen in scar tissue, which begins as dense connective tissue but undergoes metaplastic change to become calcified. Atherosclerotic plaque or blood clots (thrombi) within blood vessels can undergo metaplastic change and calcify.

Dysplasia (Figs. 13.5 and 13.6) **Dysplasia** means disordered growth and loss of the normal maturation of cells. It implies an alteration in the size, shape, and architectural organization of adult cells in tissue (oral mucosa). These changes are often considered premalignant; however, dysplasia does not necessarily progress to cancer. The specific cellular changes seen in epithelial dysplasia include the following: prominent nucleoli, nuclear pleomorphism, hyperchromatic nuclei that are abnormally large for the size of the cell, increased and

abnormal mitosis, and multinucleation. Clinically, dysplastic changes often appear as red, white, pigmented, and ulcerative lesions affecting the oral mucosa. The condition may also manifest as an abnormality of development in susceptible persons. Some examples include anhydrotic ectodermal dysplasia, cleidocranial dysplasia, and dentin dysplasia. Fibrous dysplasia is another developmental condition in which normal bone is replaced by fibrous connective tissue, causing a weakening of the bone and a distortion of its shape and size. These patients may have endocrine gland problems and café au lait brown spots on the skin.

Carcinoma (Figs. 13.7 and 13.8) **Carcinoma** is a malignant neoplasm made up of epithelial cells (skin or mucosa) that can infiltrate the surrounding tissues and give rise to metastases (distant lesions). The malignant changes seen include increased cell numbers, variable cell size and shape, and abnormal cell architecture. Carcinomas are the most common form of oral cancer and clinically may appear as red, white, pigmented, or ulcerative lesions. The prognosis (chances for survival) becomes worse as the carcinoma is located farther back in the mouth. The most common type of carcinoma is squamous cell carcinoma; other types include verrucous carcinoma, often seen on the gingiva; mucoepidermoid carcinoma, occurring in the major and accessory salivary glands; and adenocarcinomas as observed in the maxillary sinus mucous lining. Basal cell carcinomas develop in the skin of the face (sun-exposed regions) and are locally destructive, but do not metastasize.

Metastasis is a process by which carcinomas spread to distant sites generally via the lymphatics and sometimes via the bloodstream. The metastatic cells become established at distant sites—such as the liver, lungs, brain, kidneys, or jaw bones—and replace those tissues, ultimately causing them to fail functionally. Even distant metastases can be treated today by methods such as chemotherapy with cure and long-term survival. **Carcinoma in situ** is a term used to describe cancerous tissue limited to the epithelium that has not spread past the junction between the epithelium and the underlying connective tissue. For carcinomas, early recognition and biopsy followed by timely initiation of treatment can be lifesaving.

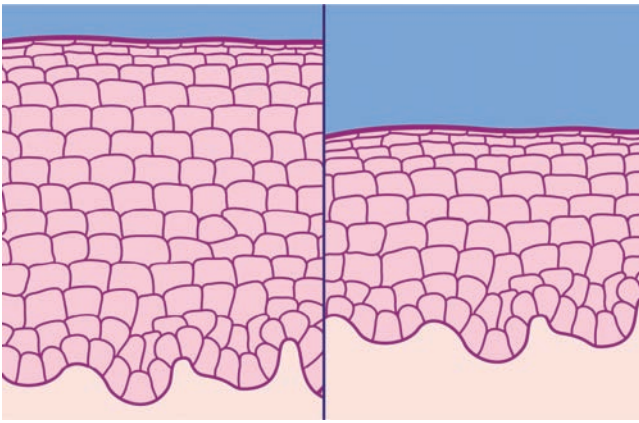


Fig. 13.1. Hyperplasia: increased number of cells (left), normal (right).

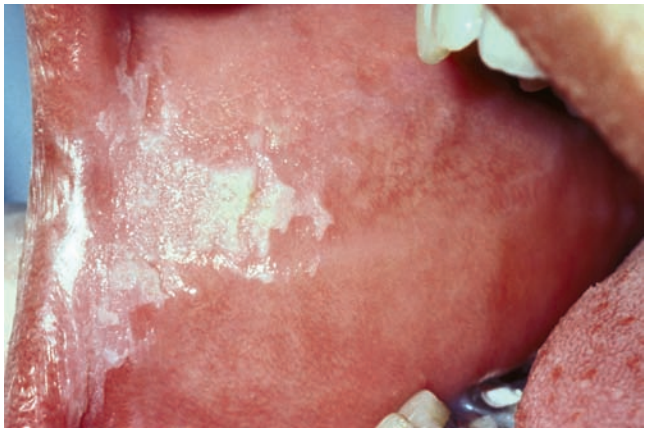


Fig. 13.2. Hyperplasia: of surface cells as a result of cheek biting.

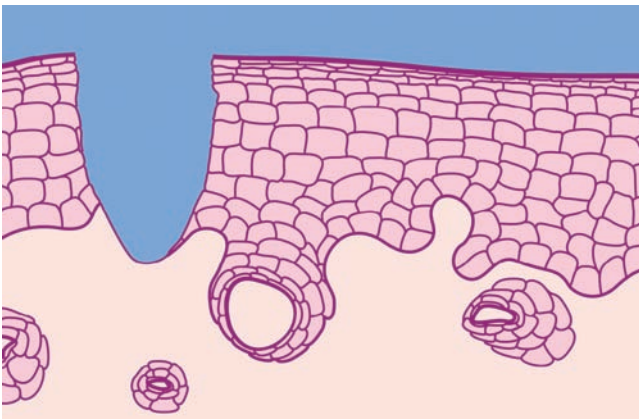


Fig. 13.3. Metaplasia: altered cell type, number, and architecture (left), normal (right).



Fig. 13.4. Sialometaplasia: minor salivary gland duct cells altered.

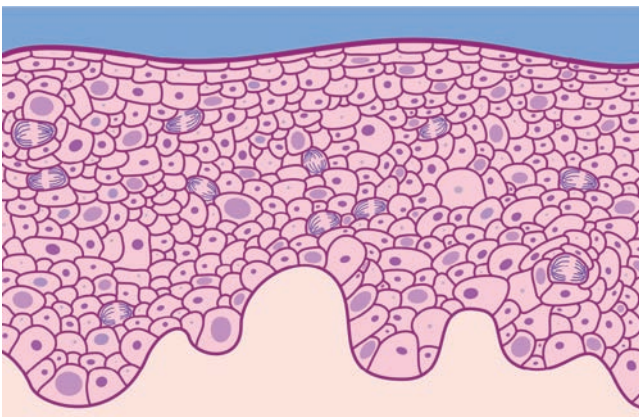


Fig. 13.5. Dysplasia: altered and premalignant cells.



Fig. 13.6. Dysplasia: leukoplakia; epithelial dysplasia of floor.

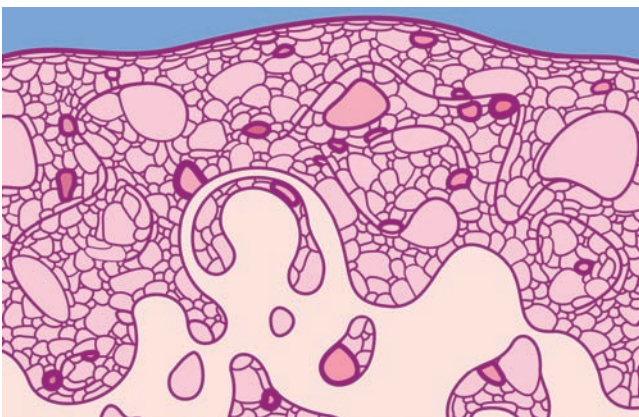


Fig. 13.7. Carcinoma: abnormal, malignant, and invasive cells.



Fig. 13.8. Carcinoma: of epithelial cells of palate and alveolar ridge

CASE STUDIES

CASE 3. (Fig. 13.9) This 53-year-old woman came to the dental clinic because of burning, painful gingiva of several months duration. An incisional biopsy was performed, and during the initial incision, the gingiva began to slough.



1. Describe the clinical findings.
2. Which term best describes this lesion?
 - A. Fissure
 - B. Vesicle
 - C. Bulla
 - D. Erosion
3. Is this a normal finding, a variant of normal, or disease?
4. How is this condition different from an ulcer?
5. Do you expect this condition to be symptomatic?
6. List conditions you should consider in the differential diagnosis, and note which condition is most likely the correct diagnosis.
7. Once this condition is brought under control, what precautions should be taken during periodontal debridement procedures?

CASE 4. (Fig. 13.10) This young lady presents to your dental office with a sore tongue. She claims the lesion appeared 3 days ago, after partying with friends late into the night. She has had similar-appearing lesions under her tongue in the past, but this is the biggest lesion she has ever had. She is taking birth control pills and no other prescription medications. Her medical history form has no positive entries for any systemic diseases.



1. Describe the clinical findings. Include mention of the size, shape, color, surface, borders, and location.
2. How can you determine the size of the lesion from the findings presented?
3. What diagnostic and descriptive term best describes this lesion?
4. Do you expect this condition to be symptomatic? Why?
5. Is this a normal finding, a variant of normal, or disease?
6. True or false: This condition can be caused by subgingival calculus.
7. List conditions you should consider in the differential diagnosis for this condition, and note which condition is most likely the correct diagnosis.
8. What clinical features would help distinguish the conditions you listed in the differential diagnosis?
9. Which of the following should be considered with respect to periodontal maintenance therapy?
 - A. It is okay to clean her teeth, as long as oral hygiene measures and food avoidance is discussed.
 - B. It is okay to clean her teeth, just provide care to the contralateral side.
 - C. Therapy should be provided in the affected quadrant because there is likely a cause-and-effect phenomenon.
 - D. Therapy should be delayed until healing occurs and the patient is more comfortable.

Oral Conditions Affecting Infants and Children

Dental Objectives:

- Define and identify common oral conditions of infants and children.
- Recognize the causes and clinical features of these conditions.
- Use the diagnostic process to distinguish similar-appearing oral anomalies of infants and children.
- Describe the consequences of disease progression with respect to these conditions.
- Recommend appropriate treatments for common oral conditions of infants and children.

Dental Hygiene Objectives:

- Define and describe the clinical appearance of common oral conditions of infants and children.
- Identify conditions discussed in this section that (1) require the attention of the dentist and/or (2) affect the delivery of periodontal debridement and oral hygiene measures.

ORAL CONDITIONS AFFECTING INFANTS AND CHILDREN

Commissural Lip Pits (Fig. 14.1) **Commissural lip pits** are dimplelike invaginations of the corner of the lips. They may be unilateral or bilateral but tend to occur on the vermilion portion of the lip. They are generally <4 mm in diameter, and exploration of the pits yields a sealed depression. Commissural lip pits represent failure of fusion of the embryonic maxillary and mandibular processes. The pits occur more frequently in males than females, and prevalence differs between adults and children. For example, lip pits have been documented in <1% of children but in up to 10% of adults. These prevalence figures suggest that the invaginations may be developmental. However, an autosomal dominant pattern of transmission (inherited pattern) has been reported in some families. No treatment is required.

Paramedian Lip Pits (Fig. 14.2) **Paramedian lip pits** are congenital depressions that occur in the mandibular lip, most often on either side of the midline. They develop when the lateral sulci of the embryonic mandibular arch fail to regress during the sixth week in utero. Paramedian lip pits are more often bilateral, symmetric depressions with raised, rounded borders. However, single pits and unilateral pits have been described. Inspection reveals a sealed depression that may express mucin. Paramedian lip pits are often inherited as an autosomal dominant trait in combination with cleft lip or cleft palate and hypodontia. When these features occur together, the condition is called **van der Woude syndrome**. The gene responsible for lip pits shows variable penetrance. That is, some patients who carry the gene may have a minor or submucosal cleft palate or no cleft yet and pass the full syndrome on to their children. Paramedian lip pits require no treatment unless they are of esthetic concern. The clinician should inquire about affected family members and examine them for cleft lip and palate.

Cleft Lip (Figs. 14.3 and 14.4) **Cleft lip** is the result of a disturbance of lip development in utero. The upper lip is most commonly affected. Cleft lip results when the medial nasal process fails to fuse with the lateral portions of the maxillary process of the first branchial (pharyngeal) arch. Fusion normally occurs during the sixth and seventh weeks of embryonic development.

Cleft lip occurs in about 1 in 900 births and more often in Asian and Native American persons than in Caucasians. The condition occurs more in males than in females and is more severe in males. About 85% of cleft lips are unilateral and nonmidline; 15% are bilateral. Midline cleft lip results from failure of fusion of the right and left medial nasal processes and is rare.

The severity of cleft lip varies. A small cleft that does not involve the nose is called an **incomplete cleft**; these sometimes appear as a small notch in the lip. A **complete cleft lip** involves the nasal structures in 45% of cases and is often associated with cleft palate. Cleft lip and palate can result from expression of abnormal

patterning genes that are transmitted by autosomal dominant and recessive inheritance, as well as X-linked inheritance. To date, there are about 400 inherited syndromes associated with cleft lip or palate. Drugs taken during pregnancy (alcohol, tobacco smoke [nicotine], anti-seizure drugs) increase the risk of clefts. Low intake of vitamin B (folic acid) is also contributory, therefore folic acid supplements are recommended during pregnancy.

Cleft Palate (Figs. 14.5–14.8) The palate develops from the primary and secondary palate. The (anterior) primary palate is formed by fusion of the right and left medial nasal processes. It is a small triangular mass that encompasses bone, connective tissue, labial and palatal epithelium, and the four incisor tooth buds. The (posterior) secondary palate is formed by fusion of the palatine processes or shelves of the maxillary process. Palatal fusion is initiated during the 8th week in utero by expansion of the mandible; this permits the tongue to drop down and allows the palatal processes to grow inward. The palatal shelves merge with the primary palate, and fusion progresses posteriorly. Except for the soft palate and uvula, fusion of the palate is generally completed by the 12th week of gestation.

Disruption in palatal fusion leads to clefting. A **cleft palate** can involve the soft palate only; the hard palate only; the hard and soft palates; or the hard and soft palates, alveolus, and lip. Cleft palate without lip involvement occurs in about 30% of cases.

Bifid uvula (Fig. 14.5) is a minor cleft of the posterior soft palate. It occurs more commonly in Asian and Native American persons; the overall incidence is about 1 in 250 people. A **submucosal palatal cleft** may occur with bifid uvula. It develops when the muscles of the soft palate are clefted but the surface mucosa is intact. The clefted region is notched. An **incomplete cleft palate** is a small break in the hard or soft palate that permits communication between the oral and nasal cavities. A **complete cleft palate** extends forward to include the incisive foramen. The triad of cleft palate, micrognathia (small jaw) and retrognathia of the mandible, and glossoptosis (posterior displacement of the tongue) is called **Pierre Robin syndrome**. This condition is characterized by respiratory and swallowing difficulties.

Cleft lip and palate are often associated with cleft alveolus, malpositioned and missing teeth (or hypodontia, most commonly the lateral incisor), and occasionally supernumerary teeth. Cleft palate causes feeding and speech impairment and prominent malocclusion. Treatment involves a multidisciplinary team consisting of a pediatrician, pediatric dentist, oral and plastic surgeon, orthodontist, and speech therapist. Surgical lip closure is generally accomplished early in the infant's life (after 18 months), whereas cleft palate may require several surgical procedures because of growth plate considerations.



Fig. 14.1. Commissural lip pits: depression at commissure.

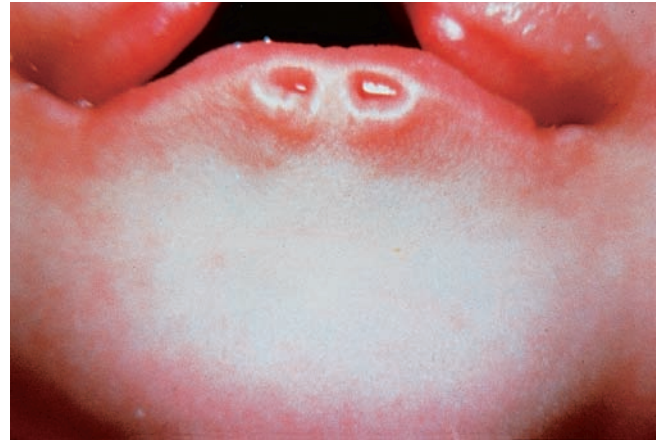


Fig. 14.2. Paramedian lip pits: with cleft lip and palate.



Fig. 14.3. Incomplete cleft lip: rare midline type.



Fig. 14.4. Bilateral cleft lip.



Fig. 14.5. Bifid uvula: mild case.



Fig. 14.6. Bifid uvula: more severe case.



Fig. 14.7. Cleft soft palate.



Fig. 14.8. Cleft lip and cleft palate.

ORAL CONDITIONS AFFECTING INFANTS AND CHILDREN

Congenital Epulis (Fig. 15.1) The **congenital epulis** is a benign, soft tissue growth arising exclusively in newborns from the edentulous alveolar ridge or palate. It develops most commonly in the anterior maxilla and is 10 times more likely to occur in females than in males. The lesion is a soft, pink fleshy growth that is compressible. The surface has prominent telangiectasis, and the base is either pedunculated (stalklike) or sessile (broad). Lesions can be several centimeters in diameter and should be excised. Recurrence is unlikely. Histologically, numerous granular cells are seen. Multiple lesions are present in 10% of cases.

Melanotic Neuroectodermal Tumor of Infancy (Fig. 15.2) This rare benign tumor typically appears as a rapidly growing mass in the anterior maxilla during the first year of life. The tumor probably arises from neural crest cells. It shows no sex predilection and begins as a small pink or red-purple nodule that resembles an eruption cyst. Radiographs usually show localized and irregular destruction of underlying alveolar bone and a primary tooth bud floating in a soft tissue mass. Urinary levels of vanilmandelic acid are elevated in conjunction with this tumor. Treatment is conservative excision. Histologic examination often shows melanin pigmentation. Recurrence and metastasis are rarely documented complications.

Dental Lamina Cysts (Fig. 15.3) The **dental lamina** is a band of epithelial tissue seen in histologic sections of a developing tooth. Remnants of the dental lamina that do not develop into a tooth bud may degenerate to form dental lamina (inclusion) cysts. These cysts are classified according to clinical location. **Gingival cysts of the newborn** are tiny keratin-filled cysts. They are often multiple and whitish and usually resolve when the tooth erupts. **Palatal cysts of the newborn** can be either Epstein's pearls or Bohn's nodules. **Epstein's pearls** arise from epithelial inclusions that become entrapped at the median palatal raphe during fusion of opposing embryonic palatal shelves. **Bohn's nodules** arise from remnants of minor salivary glands. The pearls and nodules are usually small, firm, asymptomatic, and whitish. They may occur in small groups anywhere in the hard palate. Most resolve spontaneously after several weeks, but may be incised to promote healing.

Natal Teeth (Fig. 15.4) **Natal teeth** are teeth that are present at birth or erupt within 30 days of birth. They occur in about 1 in 3,000 births. They often consist of cornified and calcific material, do not have roots, and are mobile. However, many natal teeth simply represent premature eruption of the primary teeth. The most common natal teeth are the mandibular central incisors; the mandible is affected 10 times more often than the maxilla. Natal teeth have been reported to cause ulcers of the ventral tongue (**Riga-Fede disease**) that result from irritation during nursing. In this case, smoothing of the incisal edge or extraction may be needed.

Eruption Cyst (Gingival Eruption Cyst, Eruption Hematoma) (Fig. 15.5) The **eruption cyst** is a soft tissue cyst surrounding the crown of an unerupted tooth. It is a variant of the dentigerous cyst. Children younger than 10 years of age are most commonly affected. It appears as a small, dome-shaped, translucent swelling overlying an erupting primary tooth. The cyst is lined by odontogenic epithelium and is filled with blood or serum. The presence of blood casts a red, brown, or blue-gray appearance to the cyst. No treatment is necessary because the erupting tooth eventually breaks the cystic membrane. Incising the lesion and allowing the fluid to drain can relieve symptoms.

Congenital Lymphangioma (Fig. 15.6) The **congenital lymphangioma** is a benign mass of lymphatic vessels that may present in the mouth of infants. The tongue, alveolar ridge, and labial mucosa are common locations. Less commonly, the parotid gland and floor of mouth are affected. There is a 2:1 predilection for males, and 5% of African American neonates have one or more **alveolar lymphangiomas**. A lymphangioma typically produces a swelling that is asymptomatic, compressible, and negative on diascopy. When superficial, the swelling is composed of single or multiple discrete papulonodules that may be pink or dark red-blue. Deep-seated tumors produce diffuse swellings with no alteration in tissue color unless hemorrhage occurs. Large lymphangiomas of the neck are called **cystic hygromas**. Intraoral lymphangiomas may regress spontaneously, but persistent lesions should be excised or intralesionally injected with sclerosing solution.

Thrush (Candidiasis, Moniliasis) (Fig. 15.7) Thrush, or **acute pseudomembranous candidiasis**, is a type of candidiasis (fungal infection) of mucosal membranes caused by *Candida albicans*. Lesions appear as milky-white curds on the oral mucosa, primarily the buccal mucosa, palate, and tongue. The curds are easily wiped off, leaving a red, raw, painful surface. Newborns often acquire the infection from the mother's vaginal canal during birth and show clinical signs of infection within the first few weeks of life. Fever and gastrointestinal irritation may accompany the disorder. Treatment consists of topically applied antifungal agents.

Parulis (Gum Boil) (Fig. 15.8) The **parulis** is an inflammatory response to a chronic bacterial infection of a nonvital tooth. It occurs most commonly in children when a pulpal infection spreads beyond the furcation of a posterior tooth and drains through a sinus tract to the surface. The parulis appears as a small, raised, fluctuant yellow-to-red boil that arises near the mucogingival junction of the affected tooth. Pressure to the area results in discharge of pus from the center of the lesion. Most cases are asymptomatic; however, palpation of the tooth or surrounding structures may elicit pain. The condition resolves when the infection is eliminated. Failure to eliminate the infection in a primary tooth can affect the development of the succedaneous (permanent) tooth.

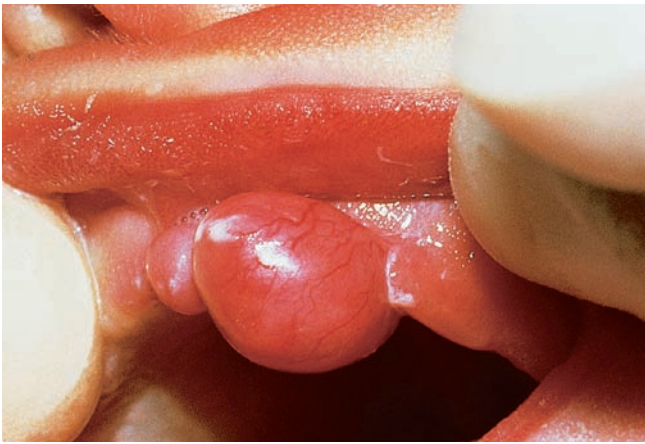


Fig. 15.1. Congenital epulis of the newborn: a pink nodule.

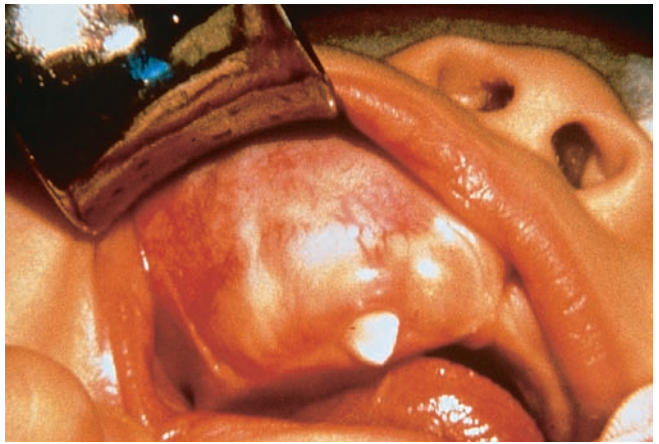


Fig. 15.2. Melanotic neuroectodermal tumor of infancy.



Fig. 15.3. Dental lamina cysts and Epstein's pearl.



Fig. 15.4. Natal teeth: mandibular incisors in a newborn.



Fig. 15.5. Eruption cyst: blue, dome-shaped cyst nodule.

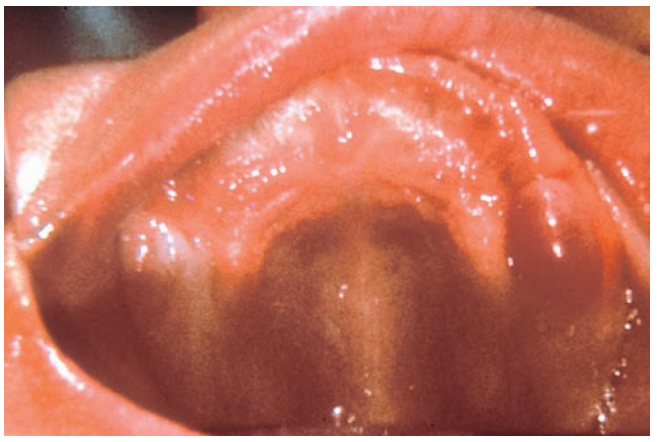


Fig. 15.6. Congenital lymphangioma.



Fig. 15.7. Thrush caused by *Candida albicans*.



Fig. 15.8. Parulis: nonvital primary first molar.

CASE STUDIES

CASE 5. (Fig. 15.9) This 26-year-old woman presents with the condition shown in the clinical image. The condition has been present since childhood. She is interested in speaking with the dentist about surgical correction.



1. Describe the clinical findings.
2. Do you expect to see similar findings in the upper lip or any defects in the upper lip?
3. Is this a normal finding, a variant of normal, or disease? And how severe is this condition?
4. What is the cause of this condition?
5. Do you expect this condition to be symptomatic?
6. List conditions you should consider in the differential diagnosis, and note which condition is most likely the correct diagnosis.
7. What are the treatment options for this condition?

CASE 6. (Fig. 15.10) A young woman brings her child to the dental office for the first time. The child has a noticeable growth within the mouth. The mother claims that the growth first presented several days ago but has been getting bigger every day. She is concerned that her child is now feeding less and this entity may be a tumor. The adjacent teeth do not seem to be affected.



1. Describe the clinical findings, and explain why it appears translucent.
2. How old is the patient?
3. Is this a normal finding, a variant of normal, or disease? And how severe is this condition?
4. What is the cause of this condition?
5. Do you expect this condition to be symptomatic?
6. List conditions you should consider in the differential diagnosis for this condition, and note which condition is most likely the correct diagnosis.
7. What are the treatment options for this condition?

Dental Anomalies

Dental Objectives:

- Define and identify common dental (tooth and root) anomalies.
- Recognize the causes and clinical features of these conditions.
- Use the diagnostic process to distinguish similar-appearing dental anomalies.
- Describe the consequences of disease progression with respect to dental anomalies.
- Recommend appropriate treatments for dental (tooth and root) anomalies.

Dental Hygiene Objectives:

- Define and describe the clinical appearance of common alterations in tooth morphology.
- Define and describe the clinical appearance of common alterations in tooth number, tooth structure and color, and tooth position.
- Define and describe the clinical and radiographic appearance of the different types of root resorption.
- Identify conditions discussed in this section that (1) require the attention of the dentist and/or (2) affect the delivery of periodontal debridement and oral hygiene measures.

In the figure legends, * denotes the same patient.

ALTERATIONS IN TOOTH MORPHOLOGY

Microdontia (Figs. 16.1 and 16.2) **Microdontia** refers to teeth that are smaller than normal. It occurs in about 1% of the population. Microdontia is usually bilateral and often associated with a familial trait (inherited) and hypodontia. It may occur as an isolated finding, a relative condition, or in a generalized pattern. The most common form occurs as an isolated finding involving one permanent tooth, usually the permanent maxillary lateral incisor. The term **peg lateral** is often used to describe this variant because the tooth is cone or peg shaped. Third molars are the second most frequently affected teeth. When microdontia occurs in a generalized pattern, it may be relative to the size of the jaws—that is, the teeth are normal in size, but the jaws are larger than normal. True **generalized microdontia** is rare and occurs when the jaws are normal in size and the actual tooth size is small. Generalized microdontia may be associated with pituitary dwarfism or an inherited syndrome. Cancer treatments (chemotherapy and radiation therapy) during tooth development are also causative. True microdonts should be distinguished from retained primary teeth.

Macrodontia (Fig. 16.3) **Macrodontia**, the opposite of microdontia, refers to an abnormal increase in tooth size. It is less common than microdontia. Macrodontia may affect one, several, or, rarely, all teeth. It is usually a relative phenomenon. It is more often seen in incisors and mandibular third molars and in a developmental condition known as **hemihypertrophy**, in which the affected side, including the teeth, is larger than the unaffected side. Single or even paired enlarged teeth are infrequently affected by **fusion** or **gemination** and may be clinically referred to as macrodonts until a more specific condition is determined from the radiographs. True **generalized macrodontia** is rare and may be seen in pituitary gigantism.

Dens Invaginatus (Dens in Dente) (Fig. 16.4) **Dens in dente** is a deep surface invagination of the crown or root lined by enamel. It is a common developmental anomaly that occurs in about 1% of the population. It is named because radiographically it resembles a tooth within a tooth. The condition develops embryologically when enamel grows inward (invaginate) into the coronal pulp chamber beginning at the lingual pit. It can extend a few or several millimeters in an apical direction. The condition is usually bilateral and may be relatively mild or severe with several invaginations in one tooth. The maxillary lateral incisors are most frequently affected, followed by the maxillary central incisors, mesiodens, cuspids, mandibular lateral incisors, and, rarely, a posterior tooth. The associated lingual pit is prone to caries, leading to early pulpitis and periapical inflammation. These pits require prophylactic placement of sealants. Radiographically, teardrop or bulb-shaped layers of enamel and possibly dentin extend apically toward or past the cemento-enamel junction. The presence of caries may not be evident; however, a periapical

radiolucency, such as a **globulomaxillary cyst**, alerts the practitioner to the loss of tooth vitality and the need for root canal therapy.

Accessory Cusps

Cusp of Carabelli (see Figs. 53.6 and 67.4) The **cusp of Carabelli** is an extra cusp located about halfway down the lingual surface of the mesiolingual cusp of maxillary molars, usually the first permanent molar. Primary molars may rarely be affected. It is usually bilateral. It is common in Caucasians and rare in Asians. There is often an associated groove between the cusp of Carabelli and the lingual surface of the tooth. This groove is prone to the development of stain and caries. When oral hygiene is fair and the diet is high in sugar, sealants are recommended.

Dens Evaginatus (Leong's Tubercle) (Fig. 16.5) **Dens evaginatus** is less common than dens invaginatus and is usually classified as an **accessory cusp**. It consists of a small dome-shaped elevation emanating from the central groove of the occlusal surface or the lingual ridge of a buccal cusp of a permanent posterior tooth. The condition occurs almost exclusively in mandibular premolars. It is especially common in persons of Asian descent. It is a feature of the **shovel-shaped incisor syndrome** common in Native Americans (see Fig. 18.8). The evaginated tubercle consists of enamel, dentin, and a slender but prominent pulp chamber. Pathologic pulp exposure may occur following fracture or attrition or iatrogenically during tooth preparation.

Protostylid (Fig. 16.6) The **protostylid** is an **accessory (extra) cusp** on the buccal surface of a tooth; the mesiobuccal cusp of a molar is most commonly affected. *Premolarization* is a term used to describe affected canines and *molarization* is used for affected premolars. An accessory cusp on molars is sometimes termed a *paramolar cusp*. The condition should be distinguished from the term **paramolar**, which is used to describe a small supernumerary tooth that develops lingual or buccal to a molar.

Talon Cusp (Figs. 16.7 and 16.8) The **talon cusp** (dens evaginatus) is a rare accessory cusp on an anterior tooth that results in a markedly enlarged lingual cingulum. It occurs more commonly in permanent maxillary incisors. Its name originates from the resemblance to a three-pronged eagle's talon. The talon cusp can interfere with occlusion. Also, the presence of a deep developmental groove can lead to lingual caries. Accordingly, the fissure should be prophylactically treated with a sealant. This condition may be an incidental finding or may be seen in association with the Rubenstein-Taybi syndrome, characterized by mental retardation, digital and facial anomalies, delayed or incomplete descent of the testes, and bone age below the 50th percentile.



Fig. 16.1. Microdontia: peg lateral incisor.



Fig. 16.2. Microdontia: radiograph of peg lateral incisor.



Fig. 16.3. Macrodontia: bilateral gemination.



Fig. 16.4. Dens invaginatus: tear or bulb-shaped.



Fig. 16.5. Accessory cusp: dens evaginatus occlusal no. 20.



Fig. 16.6. Accessory cusp: protostylid on buccal second molar.



Fig. 16.7. Accessory cusps: talon cusps on lingual of incisors.*



Fig. 16.8. Talon cusps: image of patient in 16.7.*

ALTERATIONS IN TOOTH MORPHOLOGY

Introduction (Fig. 17.1) **Fusion**, **gemination**, **twinning**, and **concrescence** are developmental anomalies explained on this page. Figure 17.1 illustrates the essential differences among these similar conditions. It is important to note that sometimes gemination and fusion are clinically indistinguishable. Questioning the patient of tooth loss as well as serial radiographs may be necessary to distinguish these conditions from supernumerary teeth.

Fusion (Figs. 17.3 and 17.5) In **fusion**, two separate tooth buds attempt to join. The fused portion usually consists of dentin and, rarely, enamel. It is reported to occur in <1% of the population and may be familial. The primary teeth are affected about five times more frequently than the permanent teeth. Bilateral presentations are about 10 times less common than unilateral examples. The incisors are the most commonly involved teeth. One distinguishing feature is the number of teeth; when the fused teeth are counted as one, then there is one fewer tooth than normal. This leads to another feature: excess interproximal space. An exception occurs when a tooth from the normal dentition fuses with an adjacent supernumerary tooth, creating the appearance of gemination. Fused teeth, like geminated teeth, have a linear groove along the labial or lingual surface and a notch at the incisal edge where the two teeth fused. Radiographically, fused teeth are more likely to have two separate pulp chambers and root canal spaces. However, variations occur, depending on the degree of fusion present. Fused primary teeth are often followed by hypodontia of the succeeding permanent teeth.

Gemination (Figs. 17.2, 17.4, and 17.5) In **gemination**, a single tooth bud attempts to divide into two teeth, but the division is incomplete. It occurs in <1% of the population, may be familial, and involves the primary teeth about five times more often than the permanent teeth. The most commonly involved teeth are the primary mandibular incisors and the permanent maxillary incisors. Bilateral gemination is rare. Clinically, the most difficult problem is to distinguish gemination and fusion. Because gemination involves a single tooth bud, the patient will have a normal number of teeth; however, the affected tooth will appear enlarged (**macrodon**), and crowding is evident. The crown may be normal in appearance, or it may have a notch at the incisal edge or a groove on the labial or lingual surface. Radiographically, a geminated tooth often has a single enlarged pulp chamber, an enlarged root, and an enlarged or bifid crown. However, other variations are possible, as seen in Figure 13.2.

Twinning (Figs. 17.6 and 20.2) **Twinning** is the complete division of a single tooth bud. The condition is considered very rare. The divided teeth are seen as completely separate with no connection to each other except that each tends to be a mirror image of the other. They are often

smaller than usual and could be described as **microdons**. When the teeth are counted, an extra tooth is present, and there may be crowding of the remaining teeth. To add to the confusion, this condition is difficult if not impossible to distinguish from the circumstance in which there are two microdons (a peg lateral and a microdontic supernumerary incisor) involving two separate tooth buds. For example, in Figure 20.2, it would be difficult to know whether the supernumerary lateral incisor is the result of twinning of a single tooth bud or whether it developed from an extra tooth bud that erupted.

Concrescence (Fig. 17.7) **Concrescence** is the union between two adjacent teeth along the root surfaces by cementum. The condition results from environmental or developmental factors after root formation is complete. Contributing factors include crowding during tooth development, inflammation due to infection, or trauma in which the interdental alveolar bone is resorbed. These conditions allow the adjacent tooth roots to become fused by the deposition of cementum between them. It may occur between two normal teeth, between a normal tooth and a supernumerary tooth, or between two supernumeraries. **True concrescence** occurs during the completion of tooth development and is seen most commonly between the second and third molars in the maxilla owing to lack of space. **Acquired concrescence** occurs after the teeth have completed development but are joined by hypercementosis associated with chronic inflammation (often pulpal inflammation of one of the teeth) in the region. Concrescence is important in tooth extraction. For example, when concrescence affects a third and second molar, extraction of the third molar can result in movement of the concrescenced second molar. It may or may not be possible to surgically separate such teeth.

Palato-Gingival Groove (Fig. 17.8) The **palato-gingival groove** is an important developmental defect and risk factor for periodontal disease. It occurs in 1% to 9% of the population, most commonly in persons of Chinese or East Indian descent, although it can be detected in other populations. The most frequently affected tooth is the maxillary lateral incisor, followed by the maxillary central incisor. The groove begins at the junction of the cingulum and one of the lateral marginal ridges, and extends onto the palatal root up to several millimeters. The groove is a frequent site of an unsuspected periodontal defect because cementum fails to cover the groove and the periodontal ligament fails to attach to the root. The periodontal defect is detected with a periodontal probe as part of the routine periodontal examination; then the groove is detected by probing the palatal aspect of the root adjacent to the defect. When present, the periodontal prognosis is diminished.

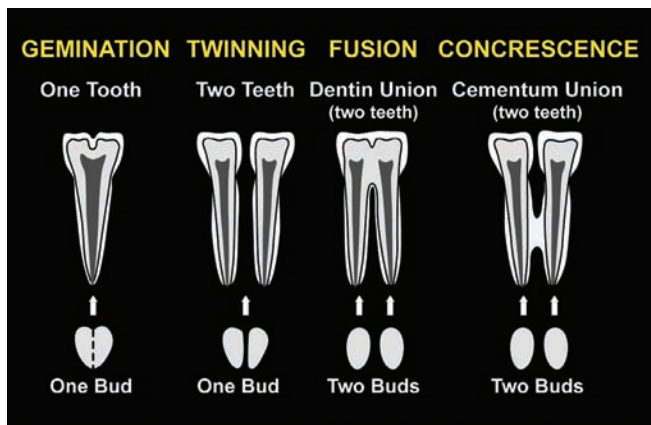


Fig. 17.1. Gemination, twinning, fusion, and concrescence.

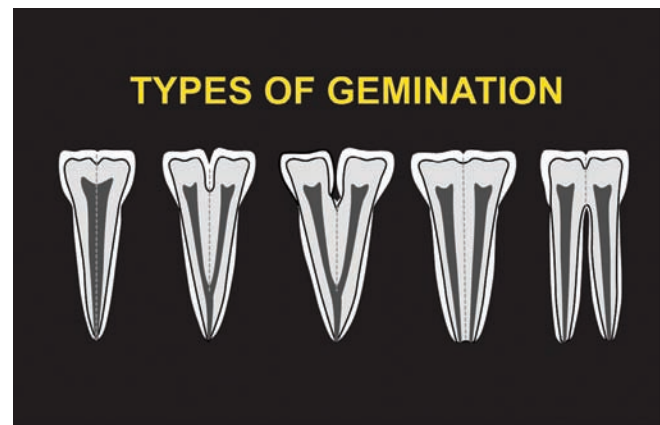


Fig. 17.2. Variants of gemination.



Fig. 17.3. Fusion: union of two teeth by dentin or enamel; bilateral.



Fig. 17.4. Gemination: a divided lower lateral incisor.

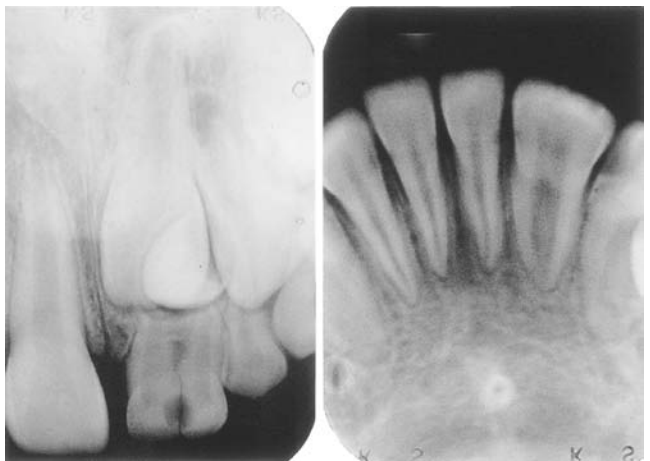


Fig. 17.5. Fusion (left), gemination (right).



Fig. 17.6. Twinning: extra tooth is a twin.



Fig. 17.7. Concrescence: Fused cementum second and third molars.



Fig. 17.8. Palato-gingival groove and associated periodontal defect.

ALTERATIONS IN TOOTH MORPHOLOGY

Supernumerary Roots (Fig. 18.1) These are developmental extra roots. Any tooth may be affected. The normal number of roots is one in incisors, canines, mandibular premolars, and maxillary second premolars; two in maxillary first premolars and mandibular molars; and three in maxillary molars. **Supernumerary roots** occur more frequently in permanent third molars, mandibular canines, and premolars. Mandibular molars are affected in 1% of Caucasians, 20% of persons of Mongolian extraction, and as many as 44% among Aleuts. Radiographically, extra roots can be suspected when the root canal space abruptly diminishes in size and bifurcates into two separate canals. This entity is important in root canal therapy, extraction, prosthetics, and orthodontics.

Ectopic Enamel: Enamel Pearl (Figs. 18.2 and 18.3) **Enamel pearls** are small pearl-like deposits of enamel at unusual locations, often at the furcation of molars. They are more common in Asians, Malaysians, and American Indians, and seven times more common in maxillary molars. In upper molars, they usually locate on the mesial or distal root surface; in mandibular molars, they occur on the buccal or lingual surface. Enamel pearls are dome shaped, 1 to 2 mm in size, and rarely multiple. They do not contain pulp tissue. They may be associated with chronic periodontal infection and may hinder periodontal instrumentation. In Figure 18.3, the pearl moves opposite to the x-ray beam location; thus, the pearl is on the palatal surface of the tooth. Radiologically, false pearls occur in lower molars (see Fig. 6.7).

Ectopic Enamel: Cervical Enamel Extensions (Fig. 18.4) **Cervical enamel extensions** occur at the midbuccal cementoenamel junction of molars and consist of a V-shaped, smooth or roughened extension of the buccal enamel extending toward the furcation area. They are more common in persons of Asian descent and are less frequent in Caucasians. Mandibular molars are involved more often than maxillary molars, with the first, second, and third molars affected in descending order of frequency. The cervical extension is difficult to see in radiographs but may be detected with an explorer or periodontal probe. Cervical enamel extensions are associated with periodontal pocket formation and furcation involvement, as well as with an inflammatory cyst known as the **buccal bifurcation cyst** (Fig. 31.7). The cyst is a radiolucent area delineated by a thin radiopaque crescent-shaped line superimposed on the buccal root surface of the tooth, sometimes extending to the distal. Cystic complications tend to develop in children and adolescents.

Dilaceration (Fig. 18.5) **Dilaceration** is a sharp bend in a root—or less frequently, the crown of a tooth—usually >20 degrees. Common causes are interference with the path of eruption as a result of crowding, trauma, adjacent bony lesions, or orthodontic traction. Dilaceration is common in third molar and maxillary lateral incisor

roots. The bend is often toward the distal, distobuccal, or distolingual. When the dilaceration is buccal or lingual, radiographically it appears as a bull's-eye, with the center representing the pulp canal. Dilaceration can complicate root canal therapy and exodontia.

Bulbous Root (Figs. 18.5 and 33.6) A **bulbous root** is a developmental variation in root morphology in which the normal apical taper is replaced by localized widening of the root. This condition is genetically determined rather than a response to local factors. The bulbous appearance is the result of increased amount of dentin—not cementum. Affected teeth are more difficult to extract.

Hypercementosis (Fig. 18.6) **Hypercementosis** is the excessive deposition of secondary cementum on the root(s) of any tooth. It occurs in association with local factors (supraeruption, apical periodontal infection, occlusal trauma) and systemic conditions (Paget disease, toxic thyroid goiter, acromegaly, pituitary gigantism). In a German study of 22,000 patients, the incidence was 2%; mandibular molars, second premolars, and first premolars were the most commonly affected in descending order of frequency; and mandibular teeth were affected twice as often as maxillary. Radiographically, the root outline is enlarged and delineated by the periodontal membrane space and lamina dura. Hypercementosis occurs more often at the apical third of the root; it makes extractions more difficult.

Taurodontism (Fig. 18.7) **Taurodontism** (bull-like teeth with large body and short legs) is a condition affecting a multirrooted tooth or teeth caused by a defective gene involved in odontogenesis. It is characterized by an elongated pulpal floor and furcation, disproportionately short roots, and a lack of constriction at the cementoenamel junction. Permanent teeth are affected more often than primary teeth and molars more than premolars. The incidence ranges from 0.5% to 5%. Severity is classified as **hypotaurodont** (mild), **mesotaurodont** (moderate), and **hypertaurodont** (severe). It occurs in association with inherited conditions including **Down, Mohr, Klinefelter, and tricho-dento-osseous syndromes**; some cases of **amelogenesis imperfecta**; and in children who receive antineoplastic treatment. Taurodonts are treated as normal, although the furcation is deeper, and the pulp space affects root canal therapy.

Shovel-Shaped Incisor Syndrome (Fig. 18.8) This inherited syndrome is seen in Native Americans, Eskimos, and Hispanics. The main features are prominent marginal (shovel-shaped) ridges, accentuated lingual pits in maxillary incisors, and markedly shortened roots, especially the premolars; dens evaginatus in the lower premolars; and class VI caries at cusp tips. Patients are prone to class III and lingual pit caries in maxillary incisors.



Fig. 18.1. Supernumerary root: first molar (left), canine (right).



Fig. 18.2. Ectopic enamel: enamel pearls at furcation.



Fig. 18.3. Enamel pearl: palatal surface and perio defect.



Fig. 18.4. Cervical enamel extension: first molar, bifurcation cyst.



Fig. 18.5. Dilaceration third molar; bulbous root first molar.



Fig. 18.6. Hypercementosis: premolars.



Fig. 18.7. Taurodontism: hypertaurodont.



Fig. 18.8. Shovel-shaped incisor syndrome.

ALTERATIONS IN TOOTH NUMBERS: HYPODONTIA

Hypodontia (Figs. 19.1–19.3) **Hypodontia** is the congenital absence of *six teeth or less* because of **agenesis** (failure to develop). Similar in meaning is the term **oligodontia**, which refers to *six or more* congenitally missing teeth. The term **anodontia** is reserved for the rare condition in which *all* teeth are absent. When teeth are missing, the patient must be carefully questioned to determine the reason for hypodontia. If teeth are missing because of extraction or lack of eruption, the terms **acquired hypodontia** and **impaction** should be used instead.

Tooth agenesis and hypodontia result from defects in genes involved in odontogenesis, including *MSX1* and *PAX9*. Hypodontia may involve either sex, any race, and the primary or permanent teeth; however, it is most common in the permanent dentition. About 5% of the population is affected; the prevalence is slightly greater in women than men. Because hypodontia is often inherited, several family members often are missing the same tooth. The most frequent congenitally missing teeth are the third molars, followed by mandibular second premolars, maxillary second premolars, and maxillary lateral incisors. Common clinical signs of hypodontia are prominent spacing between the teeth, overretained primary teeth, and microdonts. Counting the teeth and radiographic findings confirm the condition.

Cleft lip and **palate** and more than 30 syndromes are associated with congenitally missing teeth, including Böök syndrome, chondroectodermal dysplasia (Ellis van Creveld syndrome), ectodermal dysplasia, Down syndrome, Hajdu-Cheney (acro-osteolysis) syndrome, incontinentia pigmenti, otodental dysplasia, and Rieger syndrome. Radiation therapy to the head and neck of infants or children and rubella (measles) during pregnancy have also been implicated. Patients who are missing the central incisors, first molars, or several teeth and have no apparent reason for this finding should be evaluated for an inherited syndrome or one of the predisposing factors mentioned above.

Acquired Hypodontia (Fig. 19.4) **Acquired hypodontia** is the loss of teeth as a result of trauma or extractions. Sport and motor vehicle accidents are the source of most traumatic cases, whereas periodontal disease, caries, and space requirements for orthodontia contribute to dental extractions and acquired hypodontia. Tooth loss produces excess space that may result in **drifting**, **tipping**, **rotation**, and **supraeruption** of adjacent or opposing teeth. In particular, splaying and spacing of the anterior teeth is an indirect result of loss of posterior teeth because of distribution of the occlusal load onto the remaining anterior teeth. In this condition, known as **posterior bite collapse**, the single-rooted anterior teeth are less able to handle the load and tilt anteriorly when the periodontal support is poor. Acquired hypodontia can produce alterations in occlusion and appearance that require surgical, orthodontic, periodontic, and prosthodontic therapy to restore function and esthetics. When all the teeth have

been extracted, the condition is referred to as complete **edentulism**. In contrast, the term **anodontia** is used when all teeth are congenitally absent.

Ankylosis (Figs. 19.5 and 19.6) **Ankylosis** is defined by the loss of the periodontal ligament and fusion of the cementum with the alveolar bone. It is often associated with **hypodontia**. Ankylosis most commonly occurs when a primary (second) mandibular molar fails to exfoliate, as a result of a missing permanent second premolar that fails to develop and erupt. It can also affect any tooth after a trauma, transplantation, reimplantation, or chronic inflammation. An ankylosed tooth is typically in infraocclusion—submerged a few millimeters below the marginal ridges of adjacent teeth. Also, the adjacent teeth are tipped toward the ankylosed tooth, and the opposing tooth may be supraerupted. Percussion of the ankylosed tooth produces a higher pitched or dulled sound compared with adjacent teeth. Ankylosed teeth often remain in the arch for many years but may exfoliate and create an edentulous space. Exfoliation is often preceded by tooth mobility, pocket formation, and radiographic changes. Treatment can involve a crown to restore occlusion and marginal ridge height.

Ectodermal Dysplasia (Figs. 19.7 and 19.8) **Ectodermal dysplasia** is a group of more than 150 inherited diseases characterized by hypoplasia or aplasia (underdevelopment or absence) of ectodermal structures, such as the hair, nails, skin, sebaceous glands, and teeth. In its best-known **hypohidrotic form**, it demonstrates an X-linked recessive inheritance. This means that the defective gene located on the X chromosome is carried by the female and manifests clinically in the male. Less common forms of ectodermal dysplasia caused by autosomal dominant and autosomal recessive transmission have been reported in females who demonstrate fewer manifestations of the syndrome. About 1 in 50,000 persons are affected. These patients have fine, smooth dry skin; hypodontia; hypotrichosis (thin, sparse blonde hair and eyebrows); and hypohidrosis (partial or complete absence of sweat glands). Other signs include a depressed bridge of the nose, pronounced supraorbital ridges, periorbital skin with fine wrinkling and hyperpigmentation, ruddy complexion, protuberant lips, indistinct vermilion border, and varying degrees of xerostomia. Most patients have **oligodontia**, and the teeth that are present often are small, conical, and tapered. The canine is the tooth most commonly present, whereas incisors are generally missing. When present, molars show reduced coronal diameter. Anodontia sometimes occurs. Most patients do not perspire and consequently suffer from heat intolerance; in infants, this may present as a fever of undetermined origin. Treatment involves a team of health care providers, genetic counseling, the avoidance of heat, and the construction of partial dentures, full dentures, overdentures, and/or implants. Patients do well with prostheses even at a young age. However, new dentures must be reconstructed periodically as the jaws grow.

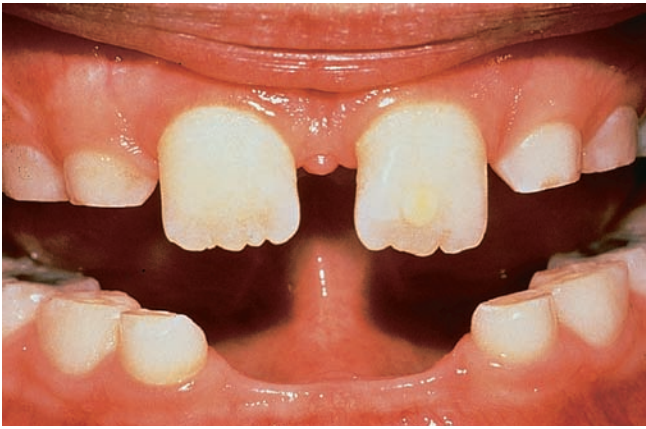


Fig. 19.1. Hypodontia: congenitally missing incisors.



Fig. 19.2. Hypodontia: congenitally missing lateral incisor.*



Fig. 19.3. Hypodontia: missing maxillary lateral incisor.*



Fig. 19.4. Acquired hypodontia: for orthodontic space.



Fig. 19.5. Ankylosis: second primary molar.



Fig. 19.6. Ankylosis: second primary molar.

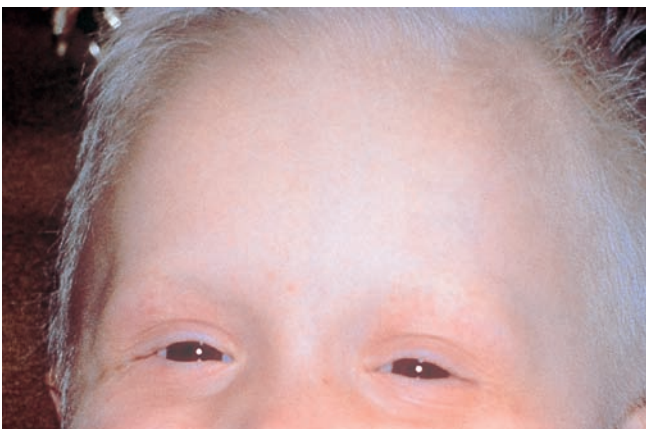


Fig. 19.7. Hypohidrotic ectodermal dysplasia: sparse hair.



Fig. 19.8. Hypohidrotic ectodermal dysplasia: hypodontia.

ALTERATIONS IN TOOTH NUMBERS: HYPERDONTIA

Hyperdontia (Figs. 20.1–20.4) **Hyperdontia** is the term for a dentition with an extra deciduous or permanent tooth; the additional tooth is termed *supernumerary*. The condition is associated with dysregulation of the RUNX family genes and focal overgrowth of the developing dental lamina. It occurs more often in the maxilla than in the mandible (8:1), more in males than females (2:1), more in permanent teeth than the primary dentition (1%–3% of the population, compared with 0.5%), and more often unilaterally than bilaterally. The most common supernumerary tooth is the **mesiodens**; this tooth is located, either erupted or impacted, near the midline between the maxillary central incisors. It may be of normal size and shape but usually is small with a short root and conically shaped crown that tapers incisally.

The second most common supernumerary tooth is the maxillary fourth molar, which can be fully developed or a microdont. When a fourth molar is buccal or lingual to the erupted third molar, the term **paramolar** is used. When the fourth molar is positioned behind the third molar, the term *distomolar* is used. Mandibular premolars are the third most common supernumerary tooth. They are usually malpositioned (ectopic eruption) because of late eruption into the arch.

Supernumerary teeth may fail to erupt or may erupt improperly. Those impacted in the jaw have the propensity to develop dentigerous cysts. Supernumerary teeth have been reported to erupt into the gingiva, palate, tuberosity, nasal cavity, and orbital rim. Space limitations in the arch often force the supernumerary tooth to fail to erupt or erupt buccally or lingually. Such teeth are often nonfunctional and may cause inflammation, food impaction, interference with tooth eruption, and esthetic and masticatory problems. In general, supernumerary teeth should be extracted to permit proper growth, development, and occlusion.

Supernumerary teeth occur as isolated occurrences but more frequently occur in several family members. The presence of several supernumerary teeth is associated with more than 15 syndromes and is commonly associated with **cleidocranial dysplasia** and **Gardner syndrome**. Inherited syndromes should be ruled out when supernumerary teeth are present.

Cleidocranial Dysplasia (Figs. 20.5 and 20.6) **Cleidocranial dysplasia** is an autosomal dominant disorder affecting the face, skull, and clavicles linked to a defect in a gene known as *RUNX2*. This gene, also known as *CBFA1*, maps to chromosome 6 and controls bone and tooth formation. Most cases are inherited; however, as many as 40% of cases result from spontaneous mutation. The syndrome affects women and men equally and is usually discovered during childhood or early adolescence.

Cleidocranial dysplasia is characterized by defective ossification of the clavicles and cranium together with oral and sometimes long bone disturbances. Prominent features include delayed closure of the frontal, parietal, and occipital fontanelles of the skull; short stature; prominent frontal eminences with bossing; small

paranasal sinuses; an underdeveloped/depressed maxilla with a high narrow palate; and relative prognathism of the mandible. The head appears large compared with the short body, the neck appears long, and the shoulders appear narrow and drooping. The clavicles may be absent or underdeveloped, permitting hypermobility of the shoulders, whereby patients can bring their shoulders together in front of the chest.

The oral changes are dramatic, particularly as seen on a panoramic image. These findings can lead to early diagnosis of the condition. The most prominent features are numerous unerupted supernumerary teeth, especially in the premolar and molar areas, and delayed eruption of the permanent teeth. The maxilla is underdeveloped, resulting in an usually high-arched, narrow, and sometimes clefted palate. There is prolonged retention of primary teeth. The permanent teeth are often short rooted and lack cellular (secondary) cementum, which may contribute to the defective eruption pattern. Treatment is complex and complicated by the fact that extraction of the primary teeth does not guarantee eruption of the secondary dentition. Surgical exposure of unerupted teeth and orthodontic and possibly prosthodontic therapy are required to produce a functional and aesthetic occlusion.

Gardner Syndrome (Figs. 20.7 and 20.8) **Gardner syndrome** is an autosomal dominant condition caused by a mutation in the adenomatous polyposis coli (*APC*) gene on chromosome 5. Although most cases are inherited, one third of cases are the result of spontaneous mutations. It affects 1 in 15,000 persons. The syndrome has prominent orofacial features characterized by hyperdontia, impacted supernumerary teeth, odontomas, and jaw osteomas that develop by puberty. In addition, patients have several epidermal cysts, dermoid tumors, and intestinal polyps. The osteomas occur most frequently in the craniofacial skeleton, especially in the mandible, mandibular angle, and paranasal sinuses. However, osteomas of the long bones are possible. Radiographs often demonstrate several supernumerary teeth, many odontomas, numerous round osteomas, and multiple diffuse enostoses that impart a cotton-wool appearance to the jaws. When superficial in the skin, these slow-growing tumors are palpable rock-hard nodules. The skin cysts (epidermoid, dermoid, or sebaceous) are smooth-surfaced lumps. Many soft tissue tumors (lipomas, fibromas, leiomyomas, desmoid tumors, and thyroid tumors/cancer) may accompany this disorder.

The most serious consideration of Gardner syndrome is the presence of multiple polyps that affect the colorectal mucosa. These intestinal polyps have an extremely high potential for malignant transformation, resulting in adenocarcinoma of the colon in 50% of patients by age 30 years and nearly 100% of patients by age 40 years. Early recognition of the orofacial manifestations necessitates prompt referral to a gastroenterologist and genetic counseling. Close annual colorectal examination is required; prophylactic colectomy is usually recommended. Facial osteoma may be surgically removed for aesthetic reasons.



Fig. 20.1. Hyperdontia: erupted mesiodens in midline.



Fig. 20.2. Hyperdontia: extra maxillary lateral incisor.

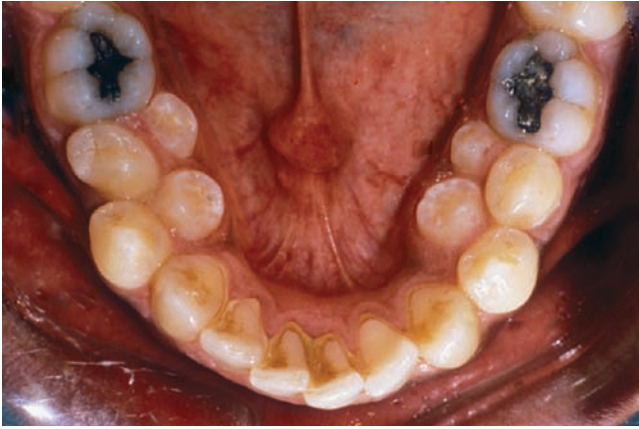


Fig. 20.3. Hyperdontia: supernumerary premolars.

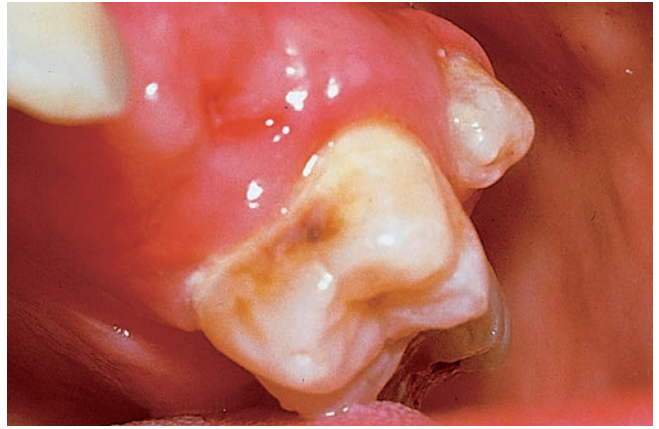


Fig. 20.4. Hyperdontia: buccal erupted paramolar.

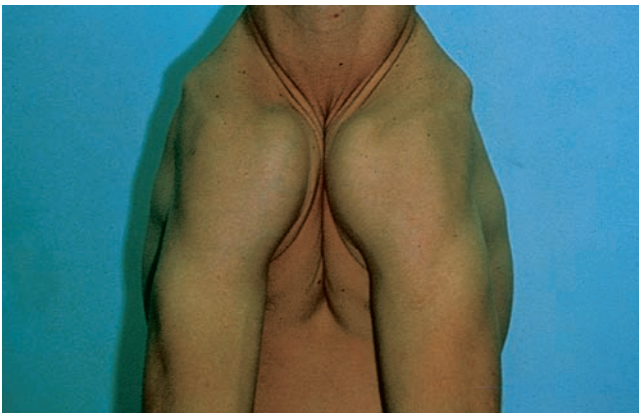


Fig. 20.5. Cleidocranial dysplasia: absent clavicles.

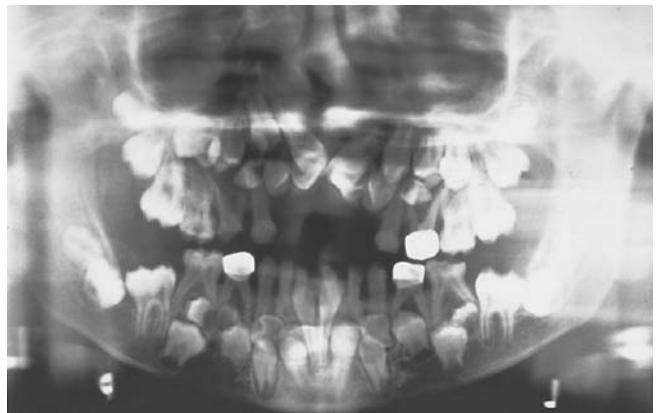


Fig. 20.6. Cleidocranial dysplasia: retained primary teeth.



Fig. 20.7. Gardner syndrome: mandibular osteomas.



Fig. 20.8. Gardner syndrome: osteomas, supernumerary teeth.

ALTERATIONS IN TOOTH STRUCTURE AND COLOR

Enamel Hypoplasia (Figs. 21.1–21.8) **Enamel hypoplasia** is incomplete or defective formation of the organic enamel matrix of primary or permanent teeth as a result of factors that affect ameloblast function. Two types exist: one caused by environmental factors, the other caused by hereditary factors termed **amelogenesis imperfecta**.

Enamel Hypoplasia: Environmental Types (Figs. 21.1–21.3) Environmental factors that can cause **enamel hypoplasia** include nutritional deficiencies of vitamins A, C, and D; infections (i.e., measles, chickenpox, scarlet fever) that result in exanthema (fever and rash); congenital syphilis; hypocalcemia; birth injury; congenital Rh hemolytic disease; local infection or trauma; ingestion of chemicals (excessive fluoride); therapeutic radiation to the jaws at a young age; and idiopathic causes. The location of the defect in the enamel dates the disturbance, as seen in Figure 21.1. Enamel formation starts incisally and proceeds cervically; thus, the defects in Figure 21.2 occurred around age 3 years.

Fever-related enamel hypoplasia occurs in all teeth undergoing mineralization during fever. Thus, maxillary and mandibular teeth are affected equally. Defects vary from a horizontal white line, pits, or a groove on the crown if the fever is brief, to severe malformations owing to lack of enamel. Mildly affected areas appear whitish; more severe cases are darker yellow to brown (Fig. 21.2).

Congenital syphilis results in clinical abnormalities such as **Hutchinson's triad**, consisting of interstitial keratitis of the cornea resulting in blurred vision; inner ear defects, resulting in hearing loss; and enamel hypoplasia of the central incisors, called **Hutchinson's incisors**. Affected incisors taper like a screwdriver with a notch on the incisal edge. Affected molars have supernumerary cusps and resemble mulberries (mulberry molars).

Turner's tooth (Fig. 21.3) is an enamel defect of a permanent tooth caused by inflammation involving, or trauma to, a primary tooth. The resulting damage insults the developing enamel of the permanent tooth. Premolars are commonly affected teeth; often the result of damage by an abscessed primary molar (Fig. 15.8). Typically, the abscess is located at the furcation immediately over the developing premolar crown. When permanent maxillary incisors are affected, common causes include abscess or trauma (from a fall) that results in intrusion of the primary incisors. Affected teeth may have a white, yellow, or brown spot or a more severe enamel defect.

Fluorosis or mottled enamel (Fig. 23.4) is caused by ingestion of excessive prescribed or natural fluoride in the drinking water during tooth development. The ideal concentration in water is about 0.7 to 1 part per million (ppm). Levels >1.5 ppm can induce **fluorosis**. The severity increases with increasing concentrations of fluoride. Mild cases of fluorosis produce chalky white areas or small pits in enamel on several teeth; moderate to severe cases produce symmetrical yellow to dark brown spots or horizontal bands in both arches. In severely affected teeth, the

enamel is soft and can fracture or wear off. Although all teeth can be affected, mandibular incisors are the least affected. Discoloration increases after eruption and can be removed by bleaching or covered with composites.

Amelogenesis Imperfecta (Figs. 21.4–21.8) **Amelogenesis imperfecta** is a group of inherited disorders characterized by a defect in one of the three stages of enamel formation (matrix formation, mineralization, and maturation). It affects the primary and permanent dentitions. The condition is divided into four main types (hypoplastic, hypomature, hypocalcified, and hypomaturation/hypoplasia with taurodontism) and 15 subtypes according to clinical, histologic, radiographic, and genetic features. Autosomal dominant forms are linked to defects in enamelin and a gene on chromosome 4. Some X-linked recessive forms exhibit mutations in the gene encoding amelogenin.

Hypoplastic amelogenesis imperfecta (type I), the most common form, is the result of deficient enamel matrix—the first stage of enamel formation. The enamel that forms is thin, well mineralized, and does not chip. There are seven subtypes referred to alphabetically as type I A through G; four are autosomal dominant, two autosomal recessive, and one X-linked dominant. Clinically, the enamel demonstrates variable patterns, including generalized or localized pinpoint pitting that is most prominent on buccal surfaces, to smooth and rough changes with white to yellow-brown tapered teeth.

Hypomaturation amelogenesis imperfecta (type II) has quantitatively normal amounts of enamel, but the matrix is immature, so the enamel is soft, discolored, and poorly mineralized; thus, a dental explorer under pressure will pit the enamel surface. In this type, the enamel appears chalky, rough, grooved, and discolored. Fracturing of the enamel is common. In its milder form, snow-capped teeth appear; in more severe cases, teeth can resemble crown preparations, with excessive interdental spacing. There are four subtypes II, A through D, with autosomal and X-linked recessive inheritance.

Hypocalcified amelogenesis imperfecta (type III) has normal enamel matrix, but undergoes no significant mineralization, resulting in a severe defect in calcification. There are two subtypes, A (autosomal dominant) and B (autosomal recessive). Developing and erupting teeth are normal in shape, with normal enamel thickness, but with unusual honey-brown color. Soon after eruption, the brown enamel undergoes severe chipping, leaving a roughened, brown dentinal surface with some enamel remaining, especially at the gingival margin. An anterior open bite is often present as a result of loss of posterior vertical dimension.

Hypomaturation/hypoplastic amelogenesis imperfecta (type IV) has predominantly hypomature or predominantly hypoplastic subtypes A and B. The teeth exhibit yellowish to opaque mottling, buccal pitting, attrition, and taurodontism. Both subtypes are seen in the **tricho-dento-osseous syndrome** (brittle nails and sclerotic bone).

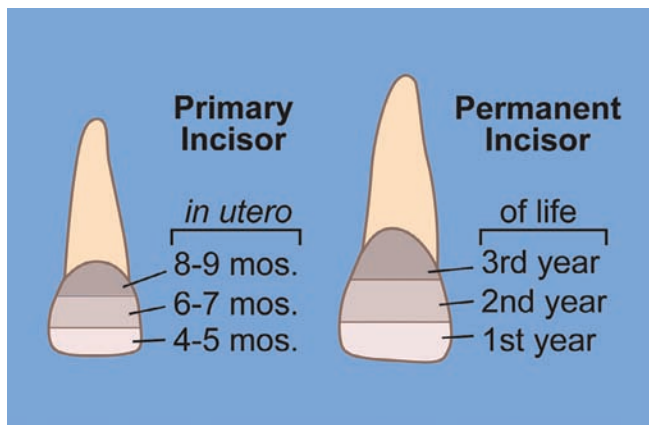


Fig. 21.1. Enamel formation in teeth.



Fig. 21.2. Enamel hypoplasia: white and brown enamel defects.



Fig. 21.3. Turner's teeth: both maxillary first premolars.



Fig. 21.4. Amelogenesis imperfecta type IIC: snow capped.



Fig. 21.5. Amelogenesis imperfecta, type ID: tapered incisors.

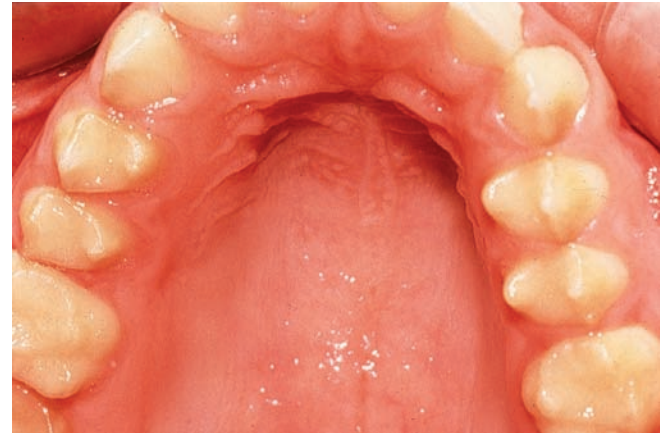


Fig. 21.6. Amelogenesis imperfecta type ID: lack of enamel.

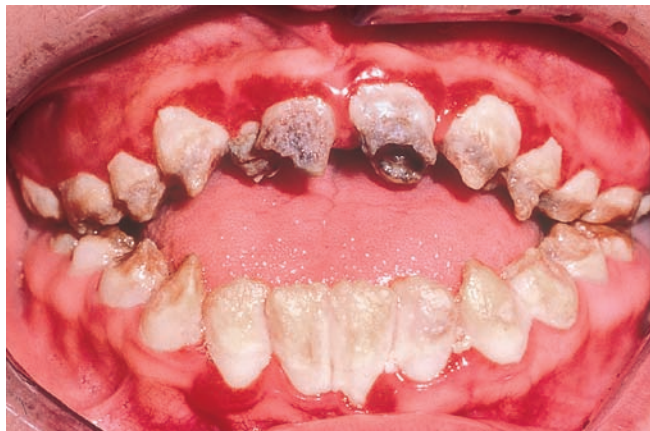


Fig. 21.7. Amelogenesis imperfecta type IIIB: open bite.*



Fig. 21.8. Amelogenesis imperfecta type IIIB.*

ALTERATIONS IN TOOTH STRUCTURE AND COLOR

Dentinogenesis Imperfecta (Hereditary Opalescent Dentin; Figs. 22.1–22.3) and Dentin Dysplasia (Figs. 22.4–22.7) These two similar autosomal dominant disorders involve abnormal dentin development of the primary and permanent teeth. For decades, they have been categorized as separate entities using the Shields classification scheme, which was based on clinical, radiographic, and histologic findings. This scheme recognizes three types of **dentinogenesis imperfecta (DI types I, II and III)** and two types of **dentin dysplasia (DD types I and II)**. Recent genetic findings, however, suggest that DD type II, DI type II, and DI type III represent increasing levels of severity of a single disease; thus, we can expect in the near future a transition from the Shields classification system to a newer genetic-based system that uses the gene defect to help classify these disorders. Until then, the Shields system is presented.

DI occurs in about 1 in 8,000 newborns. **DI Type I** is the dental feature of **osteogenesis imperfecta**, a systemic condition involving bone fragility, blue sclerae, joint laxity, and hearing impairment. It is caused by defects in the gene encoding type I collagen, which results in variable levels of defective collagen in bone and dentin. **DI type II** results from a defect in a gene that regulates dentin sialophosphoprotein (DSPP) production—the major non-collagenous proteins of dentin. **DI type II** has similar dental features as DI type I but has no bone component. **DI type III**—also known as Brandywine isolate and first described in isolated triracial groups from southern Maryland—also results from an inherited defect in DSPP production. It is described as a variable presentation of DI type II and appears clinically similar to DI types I and II, or may show slightly more severe features (see below).

All three types of DI produce misshapen and disoriented dentin tubules. The primary teeth are more severely affected than permanent teeth, with the last erupting teeth affected least. Affected teeth look clinically normal when they first erupt but shortly thereafter become discolored, amber to gray-brown or opalescent. The enamel chips and flakes away from the underlying defective dentin, resulting in fissuring and significant attrition. Radiographs often show bulbous crowns, short tapered roots, and progressive obliteration of the root canal. A less common feature is **shell teeth**, which have extremely thin dentin and dramatically enlarged pulps; this feature was first described with DI type III. DI teeth, especially primary teeth, are more susceptible to root fractures and multiple pulp exposures.

Dentin dysplasia (DD) is less common than DI. It is a rare autosomal dominant disorder characterized by dentin disorganization, pulp chamber narrowing, pulp stones, shortened roots, and numerous periapical radiolucencies. There are two types: type I radicular DD and type II coronal DD. The cause of type I is not known, but DD type II (like DI type II and type III) is caused by a defect in DSPP.

DD Type I (radicular dysplasia) primarily affects the roots of teeth, with both primary and permanent dentitions being affected. The crowns of the teeth appear normal in size, consistency, and shape, but may be slightly amber or translucent. Extremely short roots are characteristic and can cause eruption delays, tooth malalignment, mobile teeth, and early exfoliation after minor trauma. O’Carroll’s classification (**Fig. 22-4**) divides DD type I into four subgroups (a through d), depending on the degree of root shortening. In type Ia, the roots are almost nonexistent **rootless teeth (Fig. 22.5A)**. In types Ib and Ic, there is progressively more root formation; the pulp chambers are nearly obliterated and exhibit one or several thin horizontal radiolucent lines. In type Id, root length is normal; however, large pulp stones are prominent (**Fig. 22.5B**). DD types Ia, b, and c characteristically develop spontaneous periapical radiolucencies in noncarious teeth. These develop from ingress of bacteria through microscopic threads of pulpal remnants or from a periodontal defect associated with the shortened roots.

Type II (coronal) DD is less common than type I and affects the primary and permanent teeth. The primary teeth have features of DI type II (an amber, translucent appearance). Radiographically, the crowns are bulbous, the roots are thin and tapered, and there is early obliteration of the pulp spaces, exactly like DI. The permanent teeth are usually of normal shape, form, and color; however, the pulp chambers show thistle-tube deformity and pulp stones (**Fig. 22.6**). Histologically, the crowns are normal, except at the pulpal third, which has areas of globular dentin; the roots are of normal length and diameter but have pulp stones in the canal spaces and abnormal (whorls of) tubular dentin. Tooth loss is unlikely, as the roots in DD type II are of normal length and diameter and are not associated with spontaneous periapical lesions.

Regional Odontodysplasia (Ghost Teeth) (Fig. 22.8) **Regional odontodysplasia** is a rare developmental anomaly characterized by defective (thinned) enamel and dentin formation and calcifications within the pulp and dental follicle. Although most cases occur idiopathically, some are associated with syndromes and vascular or growth abnormalities. Microscopically, the dentin is interglobular and diminished in amount, the enamel is abnormal, and pulp stones are present. Clinically, ghost teeth are often carious and painful; they are usually extracted. Occasionally, they remain impacted. Affected teeth are rather radiolucent, producing a ghostlike appearance. The demarcation between enamel and dentin is absent, the pulp chambers are abnormally large, and root formation is minimal. One or more adjacent teeth can be affected, thus the term *regional*. This figure is typical and from a patient with focal dermal hypoplasia (**Goltz-Gorlin syndrome**).



Fig. 22.1. Dentinogenesis imperfecta Shields type I: enamel chipping.



Fig. 22.2. Dentinogenesis imperfecta Shields type II: amber.

4



Fig. 22.3. Dentinogenesis imperfecta: obliterated pulps.

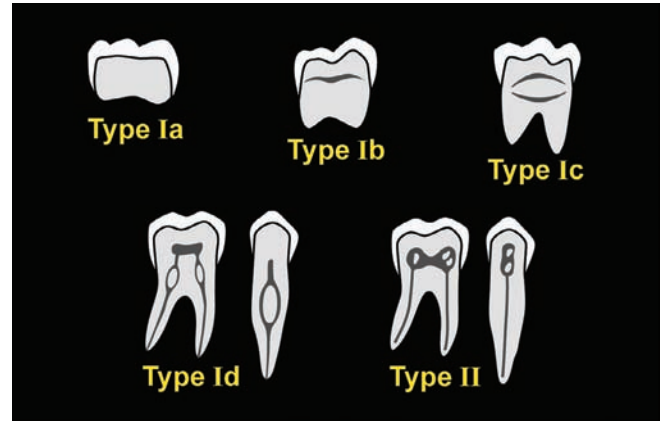


Fig. 22.4. Features of dentin dysplasia types I and II.



Fig. 22.5. Dentin dysplasia: type Ia (left); type Id (right).



Fig. 22.6. Dentin dysplasia type II: thistle-shaped pulps.*



Fig. 22.7. Dentin dysplasia type II: clinical appearance.*



Fig. 22.8. Regional odontodysplasia (ghost teeth).

ALTERATIONS IN TOOTH COLOR

Intrinsic Discoloration (Staining; Figs. 23.1–23.4) **Intrinsic staining** is a permanent change in tooth color resulting from genetic or acquired factors that interfere with odontogenesis or allow stains to be incorporated into tooth structure. Genetic processes that alter tooth structure and color include amelogenesis and dentinogenesis imperfecta and dentin dysplasia. Acquired causes of intrinsically stained teeth include restorations, trauma and infections that cause loss of tooth vitality, intake of specific drugs (such as tetracycline and ciprofloxacin) and chemicals (such as excess fluoride), and certain disease states (hepatitis, biliary disease, erythroblastosis fetalis, and porphyria) that occur during periods of tooth development. **Extrinsic staining**, in contrast to intrinsic staining, results from dark substances adhering to the external tooth surface.

Nonvital Teeth (Figs. 23.1 and 23.2) A common cause of a discolored or dark tooth is loss of vitality. In this case, the teeth are dark (yellow-brown to gray-purple) because of loss of pulpal fluids and darkening of dentin. Nonvital teeth also darken from the rupture of pulpal blood into the dentin as the result of trauma, necrosis, or infection (e.g., leprosy). If the death of the tooth occurs rapidly (within a few weeks), a pink to purple tooth can result. The typical pink discolored nonvital tooth shows greater discoloration along the neck of the crown than at the incisal edge and has been referred to as the **pink tooth of Mummery** (also see Fig. 27.6). This term arises from the fact that the mummification process results in pink teeth. Concurrent signs of nonvital teeth include caries, restorations, fractured incisal edges, or vertical fracture lines. Amalgam restorations may contribute to tooth discoloration (in which a gray-blue hue seen) either by reducing the translucency of the tooth or by uptake of metallic particles into open dentinal tubules.

Tetracycline Staining (Fig. 23.3) The tetracyclines are a group of bacteriostatic antibiotics that inhibit protein synthesis of certain bacteria. These drugs are used to treat skin and periodontal infections as well as chlamydial, certain rickettsial, and penicillin-resistant gonococcal infections. Embryos, infants, and children who receive tetracyclines are prone to develop varying degrees of permanent tooth discoloration. This is more likely to occur during long-term use and repeated short-term courses and is directly related to the dose of drug absorbed during embryogenesis and tooth development. Tetracyclines cross the placental barrier, and their presence in the bloodstream promotes deposition of the drug in the developing enamel and dentin of teeth and bones in the form of tetracycline–calcium orthophosphate. This complex causes teeth to become discolored when they erupt and are exposed to sun (i.e., ultraviolet) light. The discoloration is generalized and bandlike if the drug was administered in courses; prolonged use produces a more homogeneous appearance. The discoloration appears light yellow with oxytetracycline (Terramycin); yellow

with **tetracycline** (Achromycin); or green to dark gray with the synthetic tetracycline minocycline. Chlortetracycline (Aureomycin), which is no longer available in oral form, was best known for its ability to produce gray-brown staining. Doxycycline and oxytetracycline appear to be the least discoloring. The diagnosis is confirmed by using an ultraviolet light, which makes the teeth fluoresce bright yellow. Adults receiving long-term tetracycline therapy have been reported to acquire tetracycline staining in permanent teeth. Accordingly, alternative antibiotics should be selected in children younger than 8 years of age, and long-term tetracycline treatment should be avoided in adults if possible.

Fluorosis (Fig. 23.4) **Fluorosis** is a disturbance of the developing enamel that is caused by excess levels of fluoride in the blood and plasma. The optimal fluoride concentration in drinking water is between 0.7 and 1 ppm. At this level, fluoride is incorporated into the enamel matrix and adds hardness and caries resistance. At levels >1.2 ppm, there is increased risk for fluorosis. Blood levels are directly related to the level of fluoride ingested in water; excess levels can be acquired from drinking well water (endemic fluorosis) or from excessive water treatment. At elevated fluoride levels, ameloblasts are affected during the apposition of enamel and produce deficient organic matrix. At high levels, interference of the calcification process occurs. Mild fluorosis produces isolated, lusterless, whitish opaque spots in enamel. These spots occurring near the incisal edge and cusp tips are called **snow caps**. Moderate to severe fluorosis is characterized by generalized (symmetrically bilateral) defects ranging from several yellow to brown spots to numerous pitted enamel and mottled dark brown-white spots. In the severe form, crown morphology can be grossly altered. Maxillary incisors are more often affected than mandibular incisors.

Extrinsic Staining (Figs. 23.5–23.8) **Extrinsic stains** result from the adherence of colored material or bacteria to tooth enamel. Most extrinsic stains localize in the gingival third of the tooth above the gingival collar, where bacteria accumulate and absorb stain. **Chromogenic bacteria** produce green to brown stains in this region. These stains result from the interaction of bacteria with ferric sulfide and iron in the saliva and gingival crevicular fluid and the precipitation of chromogens into the dental pellicle. This pattern of staining is more common in children and patients with poor oral hygiene and gingivitis, where frequent bleeding from the gums results in breakdown of hemoglobin into green pigment (biliverdin). Colored fluids, such as coffee, tea, and chlorhexidine, and inhaled tobacco smoke can cause brown to black stains. These stains appear darkest in the gingival third of the tooth. Calculus and caries also discolor teeth. Calculus appears greenish black when subgingival or tan when supragingival. Caries darkens the tooth; however, unlike stains, caries causes loss of tooth structure.



Fig. 23.1. Intrinsic staining: nonvital right central.



Fig. 23.2. Intrinsic staining: pink tooth of Mummery.



Fig. 23.3. Intrinsic staining: tetracycline staining.



Fig. 23.4. Intrinsic staining: moderate to severe fluorosis.



Fig. 23.5. Extrinsic staining: chlorhexidine staining.



Fig. 23.6. Extrinsic staining: caused by chromogenic bacteria.



Fig. 23.7. Extrinsic staining: caused by tobacco and coffee.



Fig. 23.8. Extrinsic staining: applied with holiday spirit.

TOOTH ERUPTION AND ALTERATIONS IN TOOTH POSITION

Rotated Teeth (Fig. 24.1) A **rotated tooth** is one that is altered in orientation in the dental arch. The amount varies, with severe cases reversing the buccal-lingual orientation of the tooth. Rotated teeth are often associated with crowding and malocclusion.

Axial Tilting (Fig. 24.1) All teeth have a normal **axial inclination**—the long axis tilt of a tooth. Altered axial tilt is a sign of crowding, malocclusion habits or periodontal disease. For example, anterior teeth have a normal, slightly outward axial inclination, which contributes to convex lip form and appropriate arch and facial profile. Teeth may be abnormally tilted inward or outward, as occurs with the maxillary incisors in class II, division 2 malocclusion (Fig. 4.8).

Ectopic Eruption (Fig. 24.1) In **ectopic eruption**, one or more teeth erupt into an abnormal location outside the normal dental arch, usually because of lack of space. This can result in overlapping or a double row of teeth. This frequently occurs when the primary teeth (especially mandibular incisors) are retained and the permanent successors erupt lingually. Similarly, one or more supernumerary teeth may erupt adjacent to the normal teeth and compete for the location (Figs. 20.1–20.4). Other causes of ectopic eruption include cysts or tumors that force an adjacent developing tooth to erupt in an abnormal place, or syndromes (e.g., **Treacher Collins syndrome**) associated with small jaw formation. Some unusual ectopic eruption sites include the floor of the mouth, nose, sinus, and periocular region. Treatment often involves orthodontics and/or extraction.

Orthodontic Tooth Movement (Fig. 24.2) In **orthodontic tooth movement**, abnormally positioned teeth are brought into normal position. Teeth also can be moved into abnormal positions. In Figure 24.2, the first premolar has been extracted, and the canine and lateral have been positioned distally. This method can result in excellent alignment of the teeth, but may flatten the appearance of the lower face. In Figure 24.2, mild resorption of several root apices has resulted from the orthodontic forces. The thickened lamina dura and prominent periodontal membrane space are suggestive of recent orthodontic movement or possible hyperocclusion.

Transposition (Fig. 24.3) In **transposition**, two teeth exchange places, each occupying the normal place of the other within the dental arch. This may occur idiopathically or it may be a sign that a barrier to the normal path of eruption may have existed. Such patients should be examined radiographically. In this image, the maxillary canine and lateral incisor are transposed. If transposition creates an aesthetic problem, porcelain laminate veneers can remedy the situation.

Translocation (Fig. 24.4) **Translocation** occurs when one tooth erupts into an abnormal location but remains within the dental arch. In this example, the permanent

lateral incisor was congenitally missing and the permanent canine did not encounter the root of the permanent lateral incisor to guide it in, so the canine erupted into the position of the lateral incisor and the primary canine was retained. The primary canine roots can resorb and the tooth exfoliate. Alternatively, the primary canine may be retained for many years.

Distal Drift (Fig. 24.5) **Distal drift** is distal movement of an erupted tooth or teeth within the dental arch as a result of a missing tooth. The normal forces of occlusion usually cause teeth adjacent to missing teeth to drift or tip mesially. Distal drift is more prone to occur in younger persons who have had a first molar extracted. In this case, the mandibular second premolar has drifted distally into the position of the absent first molar; the first premolar and canine have drifted distally, and interdental spacing is prominent.

Migration (Fig. 24.6) **Migration** refers to the movement of an unerupted tooth into an abnormal position within the jaw. Migrated teeth usually do not erupt, but can cause root resorption of adjacent teeth. Premolars are prone to migration, and some move into the ramus and other distant sites. In our example, a second premolar has migrated to the apical region of the first molar.

Partial (Delayed) Eruption (Fig. 24.6) **Partial eruption** occurs when a tooth erupts, but not fully into occlusion. This may be caused by an impediment such as insufficient space. To assess eruption potential, root length is evaluated. For example, when root length is half complete (i.e., exceeds crown length), the tooth should be erupting through the gingiva. When the root is three-fourths formed, the tooth should be entering occlusion. When the apex has closed, eruption should be complete. Eruption potential is strongest during the first half of root development and diminishes as the apical half of root formation develops. In Figure 24.6, root length of the second premolar exceeds crown length; thus, eruption is delayed and is impeded by the first molar. Interceptive orthodontic traction and tipping can alleviate this condition.

Supraeruption (Extrusion) (Figs. 24.7 and 24.8) In **supraeruption**, one or more teeth erupt passively beyond the occlusal plane as the result of the loss of contact with an opposing tooth. Both maxillary and mandibular teeth can supraerupt, but it occurs more often with maxillary teeth. Periodontal disease may accelerate the problem, but its absence cannot prevent it. Supraeruption contributes to poor interproximal contact, food impaction, periodontal defects, and root caries (Fig. 29.6). The extruded tooth can occlude with the opposite edentulous ridge, causing pain, an ulcer, or leukoplakia, and leave little space for a replacement tooth. Extruded teeth may need root canal therapy, crown lengthening, and a crown to reestablish a normal plane of occlusion before replacing its antagonist.



Fig. 24.1. Ectopic eruption, axial tilting, and rotated teeth.



Fig. 24.2. Results of orthodontic tooth movement.



Fig. 24.3. Transposition of maxillary canine and lateral.



Fig. 24.4. Translocation of permanent canine.



Fig. 24.5. Distal drift of mandibular premolar and canine.



Fig. 24.6. Migration of premolar and delayed eruption.



Fig. 24.7. Supraeruption of mandibular second molar.



Fig. 24.8. Supraeruption of maxillary first molar.

ACQUIRED DEFECTS OF TEETH: NONCARIOUS LOSS OF TOOTH STRUCTURE

Attrition (Figs. 25.1 and 25.2) **Attrition**, considered a physiologic process, is the wearing down and loss of occlusal, incisal, and interproximal tooth structure because of chronic tooth-to-tooth frictional contact. The condition is seen most often in older adults, but the primary teeth of young children may also be affected. Attrition is often a generalized condition accelerated by **bruxism** and abnormal use of selective teeth. Flattening of the incisal and occlusal surfaces, wear facets, and widened interproximal contact areas are common findings. Occlusal examination reveals a smooth and highly polished tooth surface that is broad and angled, the outline of the dentin-enamel junction, loss of the superior interproximal space, and a receded pulp chamber. Pulp exposure is rare, however, because the deposition of secondary dentin and pulpal recession occur concurrently with attrition. Affected teeth are generally not sensitive to hot, cold, or the explorer tine, but occasionally show areas of exposed root. Restoration of the teeth may be challenging because of acquired changes in vertical dimension.

Abrasion (Figs. 25.3–25.5) **Abrasion** is the pathologic loss of tooth structure caused by abnormal and repetitive mechanical wear. Various agents can cause abrasion, but the most common form is **toothbrush abrasion**. It results from abrasive toothpastes being brushed against the teeth too frequently, with improper technique, and with too much vigor. Toothbrush abrasion produces a rounded, saucer-shaped, or V-shaped notch in the cervical portion of the facial aspect of several adjacent teeth. The abraded area is usually shiny or polished and yellow (because of exposed dentin). The dentin is typically firm and noncarious, little plaque accumulation is present, and the marginal gingiva is healthy. Premolars opposite to the dominant hand are most often affected. Abraded teeth demonstrate dentinal sensitivity to hot, cold, or the explorer tine and are more susceptible to pulp exposure and tooth fracture.

Abrasive notching of the teeth also can be created by clasps of partial dentures or by factitial injury: pins or nails habitually held with the teeth, or a pipe stem persistently clamped between the teeth. Inappropriate use of toothpicks and dental floss can also abrade the interproximal regions of teeth. Porcelain restorations placed in occlusion often result in abrasion of the incisal and occlusal surfaces of unrestored teeth in the opposite arch. When the porcelain teeth are maxillary incisors, the mandibular incisors are abraded at an upward and backward angle. Exposure to abrasive substances in the diet; chewing tobacco; powder cocaine; and long-term inhalation of sand, quartz, or silica can also promote abrasion, generally at the site of chronic exposure. For example, cocaine addiction can cause localized abrasion of the maxillary anterior teeth when the drug is chronically rubbed against the gingiva and teeth.

Abrasion is a slow and chronic process, requiring many years before signs arise. Restoration of normal

tooth contour may be unsuccessful in the long term if the patient does not alter the causative behavior.

Abfraction. **Abfraction** means *to break away* and is a term that has been used in dentistry to define the loss of tooth structure at or under the cements-enamel junction that is caused by abnormal tooth flexure. The condition is controversial, and current evidence suggests that it exists only as a hypothetical component of **cervical wear**. Past theories have suggested that the condition arises from eccentric occlusal loading, which creates flexure and shearing stresses, and disruption of the bond between enamel and dentin. The defects have been described as a sharp, wedge-shaped loss of enamel and dentin along the cervical region of the facial aspect of a tooth, most commonly mandibular premolars. Now that the term *abfraction* has fallen into disfavor, dental health care workers should examine patients closely for other causes of this unique aspect of cervical wear.

Erosion (Figs. 25.6–25.8) **Erosion** refers to the loss of tooth structure caused by chemicals such as dietary, gastric, or environmental acids that are placed in prolonged contact with the teeth. The condition is exacerbated by hyposalivation and drugs that produce **xerostomia**, because loss of saliva reduces the buffering capacity of the oral cavity. The most commonly affected tooth surfaces are the labial and buccal surfaces.

The pattern of tooth erosion often indicates the causative agent or a particular habit. For example, sucking on lemons (citric acid) produces characteristic changes of the facial surfaces of the maxillary incisors. Horizontal ridges appear initially, followed by smooth, cupped-out, yellowish depressions. Eventually, the incisal edges thin and fracture. A similar erosive pattern may be seen in dedicated swimmers whose anterior teeth are chronically exposed to chlorinated swimming pools.

Erosion limited to the lingual surfaces of the maxillary teeth (**perimolysis**, also known as **perimylolysis**) indicates chronic regurgitation or vomiting caused by **bulimia**, **anorexia**, pregnancy, hiatal hernia, gastroesophageal reflux, or alcohol abuse. Affected posterior teeth have margins of occlusal amalgams raised above the eroded and adjacent enamel. Sensitivity of the exposed area is an early symptom. Excessive consumption of sweetened beverages and carbonic acid-containing beverages may accelerate the condition. Fluoride treatments for early erosions and restorations that cover exposed dentin for more extensive lesions are treatments of choice. Elimination of the causative habit or behavior modification is required for success. In cases of gastroesophageal reflux, the condition can be controlled by medications (antacids, H₂ histamine blockers, proton pump inhibitors) and use of sodium bicarbonate rinses after episodes of reflux.



Fig. 25.1. Attrition: dentin exposure, enamel thinning.



Fig. 25.2. Attrition: polished incisors; abrasion at cervical.



Fig. 25.3. Abrasion: worn by friction against porcelain.



Fig. 25.4. Toothbrush abrasion: V-shaped grooves at cervical.



Fig. 25.5. Abrasion: from rubbing cocaine on gingiva.



Fig. 25.6. Erosion: caused by chronic lemon sucking.



Fig. 25.7. Erosion: caused by intake of carbonated beverages.



Fig. 25.8. Erosion: caused by chronic vomiting in bulimia.

ALTERATION IN ROOT STRUCTURE

Resorption (Figs. 26.1–27.8) Resorption is the loss of dental hard tissues, and it generally affects the root. The loss is caused by multinucleated dentinoclasts (cells similar to **osteoclasts**) that arise from within the pulp (i.e., internal resorption) or the periodontal ligament (external resorption). **Internal resorption** starts at the pulp-dentin interface and extends outward, destroying dentin. **External resorption** is initiated at the outer surface of the tooth, destroying cementum first, then extending inward through dentin toward the pulp. Resorption occurs most often after trauma and injury, but is also associated with inflammatory, hormonal, and infection processes. Cytokines such as interleukin-1 β and tumor necrosis factor are involved. Radiographs are generally required for the diagnosis, with the clinical diagnosis most easily determined with 3-D cone beam computed tomography. From a clinical perspective, resorption is classified as coronal, cervical, radicular, and periapical.

False Resorption (Figs. 26.1–26.3) Several conditions resemble resorption and are termed *false resorption*. An **apicoectomy** (Fig. 26.1A) is one example. In this case, the apical tooth structure has been surgically removed, not resorbed, to treat a nonvital tooth. The apex of a tooth following an apicoectomy resembles periapical root resorption; however, suggestive clues include the linear root apex appearance with a gutta-percha root canal filling or amalgam apical seal. After an apicoectomy, a periapical radiolucency representing periapical fibrous scar tissue may persist.

A second example of **false external root resorption** (Fig. 26.1B) shows loss of cementum and dentin along the lateral surfaces of the roots in the cervical regions. This type of false resorption is iatrogenic (i.e., caused by the dentist, hygienist, or self-induced injury by the patient). In this case, the patient severely damaged her teeth from overzealous use of a curette, resulting in nonvitality of a central incisor.

Another example of false resorption is the radiographic appearance of shortened roots because of technique error (i.e., overangulation when taking maxillary molar and premolar periapical radiographs). This condition, shown in **Figure 26.2**, produces a periodontal ligament space and a ghostlike root.

A fourth example of **false resorption** occurs in newly erupted teeth as shown in **Fig. 26.3**. The teeth shown in the radiograph have enlarged pulp and root canal spaces. In the first mandibular molar, the enlarged unusual (round) radiolucency is pulp tissue extending coronally that can be mistakenly diagnosed as internal resorption. In young patients, these teeth are vital upon pulp testing. However, if devitalized, the tooth will appear to have internal resorption (see below).

External Resorption (Figs. 26.4–27.4) **External resorption** is much more common than internal resorption, and the

causative factors that initiate the process are much more varied. External resorption of primary tooth roots occurs physiologically both in association with the eruption of the permanent successors and with retained primary teeth with no permanent successor. The most common causes of external resorption include pressure on the tooth from various sources, such as excessive orthodontic or occlusal forces; tumors, cysts, and impacted teeth; following trauma or reimplantation of teeth; internal bleaching; inflammation of the pulp or periodontium; and idiopathic (when no obvious cause is present).

Eruption-Related External Root Resorption (Figs. 26.4–26.6)

There are three types of resorption related to eruption. The first is **physiologic resorption**, seen in the roots of primary teeth immediately adjacent to erupting permanent teeth (**Fig. 26.4**). The second type is termed **ectopic resorption** (Fig 26.5) and is a form of **cervical resorption**. It is usually seen on the distal cervical surface of the second primary molar adjacent to an erupting permanent first molar. It is also seen on the distal cervical surface of the permanent lateral incisor intimately associated with an erupting permanent canine. Both of these are signs of crowding of the permanent teeth. The third type is termed **retained primary tooth-related root resorption** (Fig. 26.6) and is seen on the root of any retained primary tooth with no permanent successor.

Inflammatory External Resorption (Figs. 26.7 and 26.8) Many inflammatory and infectious conditions can resorb dentin and replace it with inflamed granulation tissue. This occurs commonly with long-standing pulpitis and can continue as long as the pulp remains vital. The pattern of destruction is dependent on the source of the inflammation. **Periapical root resorption** is often associated with caries that leads to an inflamed pulp. Tumors and cysts can also produce inflammatory periapical root resorption (Figs. 32.1, 32.4, 32.6, 32.8). One of the more common cysts to cause resorption is the dentigerous cyst. This cyst is slow growing, which allows it to apply slow, consistent pressure on a tooth root. In contrast, fast-growing malignant tumors often will push or go around teeth. Certain patterns of resorption with other signs may suggest the diagnosis. For example, a knife-edge pattern of root resorption is associated with ameloblastomas (Fig. 32.6), and a spiked pattern is often seen with chondro- and osteosarcomas.

External root resorption can also be solitary and idiopathic. In the example shown in **Fig. 26.8**, the distal root of the mandibular second molar is resorbed. This may have been due to unknown reasons (idiopathic) or to pressure from the adjacent and partially impacted mandibular third molar during its eruption. Distal root apices of mandibular second molars are common locations for this type of resorption.



Fig. 26.1. False resorption: A: apicoectomy; B: curette damage by patient.



Fig. 26.2. False resorption: rootless PDL space distal no. 12; overangulation.

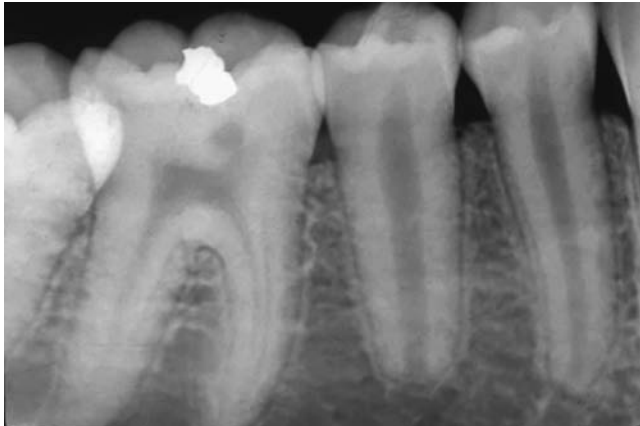


Fig. 26.3. False resorption: internal radiolucency; newly erupted molar.



Fig. 26.4. External resorption: physiologic with tooth eruption.



Fig. 26.5. External resorption: ectopic, cervical; sign of crowding.



Fig. 26.6. External resorption: missing premolar, ankylosed molar.



Fig. 26.7. External resorption: because of chronic periapical infection.



Fig. 26.8. External resorption: distal root no. 18 affected.

ALTERATION IN ROOT STRUCTURE

Orthodontic External Root Resorption (Fig. 27.1) **Orthodontic root resorption** is common when excess pressure is exerted on the teeth being moved. The condition is often generalized but limited to the teeth receiving treatment. Affected roots are shortened and flattened, and may have the root apex shifted slightly mesially or distally. In some cases, the root apices are short and rounded. Certain syndromes (i.e., shovel-shaped incisor syndrome, Fig. 18.8) can resemble generalized orthodontic root resorption, especially in the premolar teeth. In such patients with pre-existing short roots, care must be taken not to generate excessive orthodontic forces on the teeth. Orthodontic root resorption is less likely when bone hardening drugs, such as fluoride, bisphosphonates, or tetracyclines have been ingested.

External Root Resorption in Reimplanted and Transplanted Teeth (Fig. 27.2) When a tooth is completely avulsed as a result of trauma, the tooth is often devitalized, filled with root canal material, cleaned, **reimplanted** into the socket, and splinted to the adjacent teeth for stability. These cases often result in root resorption of the reimplanted tooth. In this case, only the gutta-percha root canal filling remains, and bone has replaced the root. As this is a predictable outcome, nowadays many such cases are restored with an implant.

Occasionally developing third molars are **transplanted** to replace a mandibular first molar—often the first tooth to be lost in caries-susceptible teenagers. If the pulp remains vital, the transplant can be successfully grafted. However, if vitality of the transplanted tooth is lost, the graft will fail, and the roots of the transplanted tooth are prone to resorb.

External Coronal Resorption (Fig. 27.3) This variant type of resorption occurs most commonly in the crowns of impacted or unerupted teeth and with time spreads to other parts of the tooth. The exact cause of resorption remains unknown. It occurs most frequently in impacted maxillary canines and impacted third molars, and is also seen in supernumerary teeth, especially the maxillary mesiodens. **External coronal resorption** is most easily identified when the involved tooth is completely embedded in the jaw bone, the affected tooth appears ankylosed, and there is partial or complete destruction of the tooth crown. The condition may appear similar to carious destruction and to what was previously thought to be pre-eruptive caries.

Cervical Resorption (Fig. 27.4) **Cervical resorption** is a type of external root resorption that is occasionally encountered. It begins on the external surface of the tooth in the cervical area without apparent cause. The resorption spreads coronally, resulting in a pink- or reddish-appearing crown because of enlarging vascular tissue within the crown. One or more teeth may be affected. This resorptive pattern is prone to be rapid and is also known as **invasive cervical resorption**.

Multiple Cervical Resorption of Hyperparathyroidism (Fig. 27.5) **Multiple idiopathic internal or external resorption** is a rare condition. The resorption may begin at the cervical region or in the apical region and may involve many or almost all of the teeth. Our example of this condition is a case reported by Hutton in 1985. The patient was a 22-year-old woman with secondary hyperparathyroidism associated with end-stage renal disease. Multiple teeth show idiopathic internal resorption in the cervical regions. The radiograph on the right shows the patient 4 years after the initiation of hemodialysis. The areas of root resorption have almost completely filled in, presumably with an osteodentin or cementoid tissue. It is not known if the process began externally or internally.

Internal Coronal Resorption (Fig. 27.6) **Internal resorption** is usually initiated by chronic hyperplastic pulpitis; thus, pulp testing may produce variable results. There are two radiographic patterns of internal resorption: **inflammatory and replacement or (metaplastic) internal resorption**. The pink tooth of Mummery can be the result of coronal internal resorption, which occurs when inflammation produces a hyperplastic pulp that thins the internal elements of the crown, resulting in a pink appearance (Fig. 19.2). Clinically, such teeth are weak and need endodontic therapy and a post and core. **Coronal internal resorption** may also be initiated by internal crown bleaching procedures done within endodontically treated teeth.

Inflammatory Internal Root Resorption (Fig. 27.7) **Inflammatory internal root resorption** involves a focal area of resorption within the root canal space. The pattern is distinctive. In radiographs, one sees a focal radiolucent enlargement within the root canal space that expands into the dentinal walls. The radiolucent area is typically round or ovoid and balloons beyond the pulp chamber space. Because the periapical image is two dimensional, taking cone-beam computed tomography helps determine if the resorptive process has perforated the outside margin of the root. Such perforations are often considered unre-storable.

Replacement/Metaplastic Internal Root Resorption (Fig. 27.8) **Replacement internal root resorption** involves the replacement of hyperplastic inflammatory pulp tissue with so-called osteodentin or cementumlike material. This results in an area of resorption within the root canal space that becomes diffusely radiopaque or ground-glasslike in appearance. The density of the metaplastic tissue is slightly different from that of the unaffected dentin surrounding it. Depending on the degree of replacement, the radiolucent root canal space and/or radiolucent resorptive area can produce a blurry radiolucent image because of the deposition of osseous replacement tissue around the pulp chamber.



Fig 27.1. External resorption: orthodontics; multiple teeth affected.



Fig. 27.2. External resorption: complete resorption of reimplanted incisor.



Fig 27.3. External coronal resorption: affecting an unerupted mesiodens.



Fig. 27.4. Cervical resorption: invasive type, mandibular first molar.

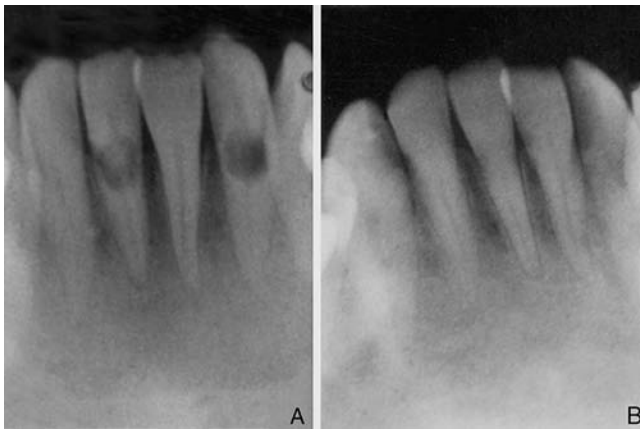


Fig 27.5. Internal resorption: A: secondary hyperparathyroidism; B: healed.



Fig. 27.6. Internal resorption: coronal; pink tooth of Mummery.

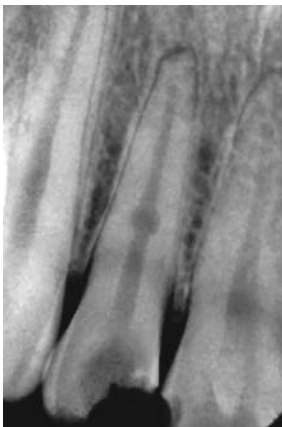


Fig 27.7. Internal resorption: inflammatory; note apical signs.



Fig. 27.8. Internal resorption: metaplastic/replacement pattern.

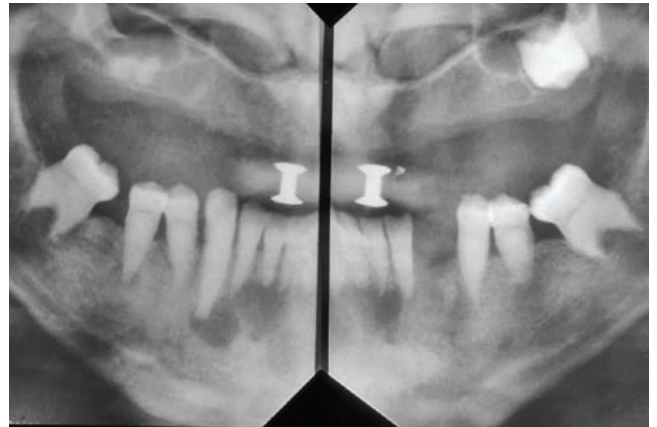
CASE STUDIES

CASE 7. (Fig. 27.9) This 22-year-old woman presents with the condition shown in the clinical image. The condition has been present since she can remember but seems to be getting worse. She is interested in cosmetic dentistry.



1. Describe the clinical findings in this quadrant.
2. What radiographic findings of this condition would you expect to see?
3. Is this a normal finding, a variant of normal, or disease? And how severe is this condition?
4. What is the cause of this condition?
5. Do you expect this condition to be painful, and if so, why?
6. List conditions you should consider in the differential diagnosis for this condition, and note which condition is most likely the correct diagnosis.
7. What are the treatment options for this condition?

CASE 8. (Fig. 27.10) This 31-year-old man recently moved to your town and presents with the condition shown in the panoramic radiograph. He is interested in having his teeth cleaned and a new upper denture made. He is concerned about a couple of lower teeth that seem loose.



1. Describe the radiographic findings shown in this panoramic image. Divide your findings into tooth-related changes and bone-related changes, and note what seems unusual.
2. What most likely caused him to lose his maxillary teeth?
3. What color and contour changes do you expect to see during the clinical examination of the mandibular teeth?
4. Is this a normal finding, a variant of normal, or disease?
5. Do you expect this condition to be painful, and if so, why?
6. List three conditions that you should consider in the differential diagnosis for this condition, and note which condition is most likely the correct diagnosis.
7. What are the treatment options for this patient?

Dental Caries

Dental Objectives:

- Define the different types of caries and their sequelae.
- Recognize the causes and clinical features of the different types of caries and their sequelae.
- Use the diagnostic process to distinguish caries, pulp polyp, and apical pathoses from similar-appearing oral conditions.
- Recommend appropriate treatments for the different types of caries and their sequelae.

Dental Hygiene Objectives:

- Identify the features of caries and their common locations.
- Define, recognize, and compare the different types of caries and the consequences of caries progression.
- Identify factors that make patients susceptible to caries.
- Discuss caries prevention with a patient who has high risk for caries.
- Identify conditions discussed in this section that (1) require the attention of the dentist and/or (2) affect the delivery of periodontal debridement and oral hygiene measures.

In the figure legends, ** denotes the same patient.

DENTAL CARIES

Caries (Figs. 28.1–28.8) Dental caries, or tooth decay, is a bacterial infection that damages the structures of teeth. The damage-caused demineralization and destruction of the organic matrix of teeth results from the interaction of acid-producing bacteria (*Streptococcus mutans*, *Actinomyces viscosus*, *Lactobacillus species*, and *Streptococcus sanguis*) in plaque with food substrates over a period of time. The bacteria produce lactic acid, which causes electrochemical changes and the outflow of calcium and phosphate ions from the mineralized portion of the tooth.

Caries begins as enamel decalcification that appears as a chalky white spot, line, or fissure. The initial lesion is termed **incipient**. As the lesion matures, it causes destruction of enamel and lateral spread along the **dentinoenamel junction (DEJ)**, through the dentin, and eventually toward the pulp. The classic features of a carious lesion are (1) color change (chalky white, brown, or black discoloration), (2) loss of hard tissue (cavitation), and (3) stickiness to the explorer tine. The color change is caused by decalcification of enamel, exposure of dentin, and demineralization and staining of dentin. Classic symptoms of caries are sensitivity to sweets, hot, and cold. These symptoms are generally absent with incipient lesions. Larger lesions permit ingress of fluids into exposed dentinal tubules. The hydrostatic (pressure) changes are sensed by pulpal nerves that transmit signals to the trigeminal sensory complex, which results in the perception of pain.

Two types of caries are classified according to location: fissural and smooth surface. **Fissural caries** is the most common form. It occurs most often in deep fissures in the chewing surfaces of the posterior teeth. **Smooth surface caries** occurs at places that are protected from plaque removal, such as just below the interproximal contact, at the gingival margin, and along the root surface. Caries is subdivided into six classes according to anatomic location. Class I caries is fissural; the remaining five classes are smooth surface caries.

Management of caries is most effective when risk factors (i.e., plaque; diet; number of initial, prior, and active caries; number of restorations; level of fluoride exposure and oral hygiene; patient compliance; number of exposed root surfaces; and salivary flow) are assessed, plaque is reduced, cariogenic bacteria are eliminated, tooth remineralization is enhanced, and teeth are repaired based on lesion size, location, and esthetic requirements.

Class I Caries (Figs. 28.1 and 28.2) **Class I caries** is decay affecting the occlusal surface of a posterior tooth. It arises when bacteria invade a central pit, deep occlusal groove, or fissure, remain sheltered for months, and produce acid dissolution of enamel. Destruction of enamel and dentin permits the carious groove to enlarge, darken, and become soft. A small class I carious lesion is the size of the tip of a sharpened lead pencil and may

exist beneath a stained fissure. Larger lesions can encompass the entire occlusal surface, leaving only a shell of facial or lingual enamel and a symptomatic tooth. Current recommendations are to detect these lesions by visual inspection, bitewing radiographs (for a radiolucent shadow in the dentin beneath the occlusal enamel), and minimal examination with an explorer. Class I caries that are incipient or small are managed by remineralization with fluoride varnish application and sealants. Larger lesions require use of composite materials or amalgam.

Class II Caries (Figs. 28.3 and 28.4) **Class II caries** is decay that affects the interproximal surface of a posterior tooth. These lesions are often difficult to identify clinically and require an astute eye, a clean, dry tooth surface, and a bitewing radiograph. One feature that may help in the detection of class II caries is the appearance of decalcification (chalkiness or translucency) along the marginal ridge that is caused by hollowing of the subjacent dentin. The caries can occasionally be seen from the lingual or buccal aspect of the interproximal contact. Class II caries is most easily recognized on bitewing radiographs. The carious lesion appears as a triangular radiolucency in the enamel just below the contact point. The base of the triangle parallels the external aspect of the tooth, and the tip of the triangle points inward toward the dentin. As the lesion reaches dentin, it spreads along the DEJ and progresses toward the pulp.

Remineralization procedures (i.e., fluoride varnish applied to demineralized areas) are used for smaller lesions limited to enamel. In moderate caries (radiographic evidence of enamel penetration along the DEJ without further penetration into the dentin), remineralization can be used if risk factors are minimal or reduced and lesions are closely monitored. Custom-made fluoride trays and daily fluoride applications reduce the risk of caries progression. Moderate to large lesions are restored with posterior composites, amalgam, or metal castings.

Class III Caries (Figs. 28.5–28.8) **Class III caries** is decay affecting the interproximal surface of an anterior tooth. Like class II interproximal caries, class III caries begins just below the contact point. Invasion results in triangular destruction of enamel and lateral spread into dentin. Interproximal caries (classes II and III) are common in persons who seldom floss their teeth and frequently consume sugar in beverages and candy. Class III caries is seen frequently in Asian and Native American persons who have prominent marginal ridges (**shovel-shaped incisors**). It can be diagnosed using transillumination (Fig. 28.6). This technique demonstrates caries as dark regions within the enamel (or deeper) located below the interproximal contact point. Class III caries in the mandibular incisors indicates high caries risk behavior.

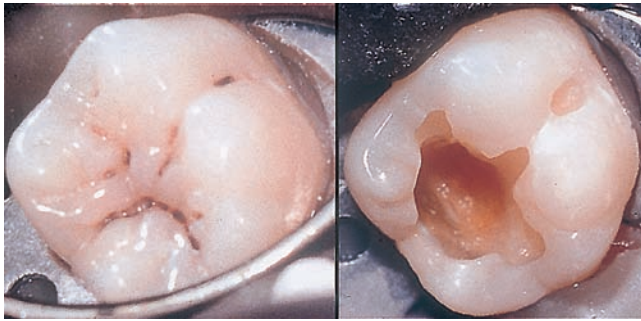


Fig. 28.1. Class I caries: before and after preparation.



Fig. 28.2. Class I caries: below occlusal enamel first molar.



Fig. 28.3. Caries: Class II mesial premolars; Class III lateral.*

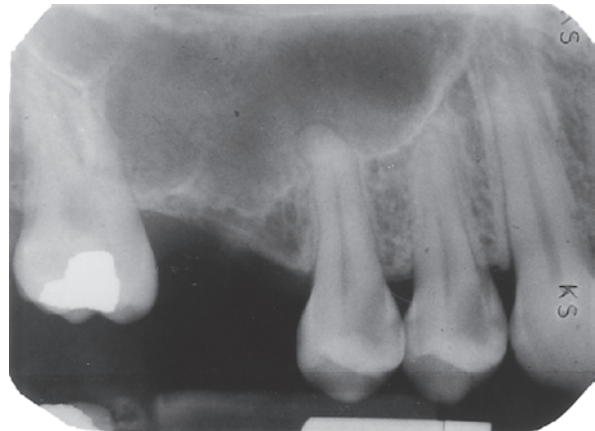


Fig. 28.4. Radiographic evidence of class II caries.*



Fig. 28.5. Class III caries: between central and lateral.†

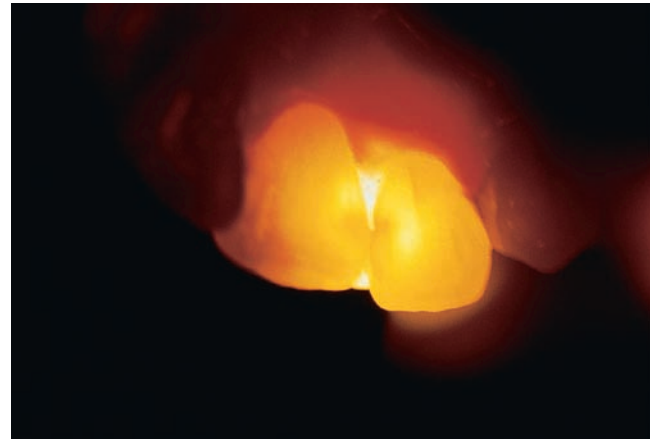


Fig. 28.6. Transillumination: demonstrates class III caries.†



Fig. 28.7. Class III caries: centrals, laterals, and canines.



Fig. 28.8. Class III caries: nos. 7 and 9; amputation caries no. 8.

DENTAL CARIES

Class IV Caries (Figs. 29.1 and 29.2) **Class IV caries** affects the interproximal surface and incisal line angle of an anterior tooth. It usually results when class III caries remains untreated, allowing the lesion to progress and undermine the dentin that supports the incisal line angle. As a result, loss of the enamel at the line angle occurs when the undermined enamel is traumatized by occlusion or chewing. Class IV carious lesions are restored with bonded composite resins, providing excellent esthetics. Porcelain laminate veneers are also useful in select cases. Restorations have longer life spans when patients are educated about the causes of caries and make changes in behavior, and occlusal forces are minimized.

Class V Caries (Figs. 29.3 and 29.4) **Class V caries** is characterized by decay along the gingival margin of a posterior or anterior tooth. Early signs of class V caries are chalky, white decalcification lines along the cervical portion of the tooth parallel to and just above the gingiva. Any plaque covering the lesion must be removed for adequate detection of the caries. With time, lesions enlarge mesially and distally at a fast rate, thereby producing an oval defect. As class V caries reach interproximal regions, spread toward the contact point occurs, and the lesion becomes L-shaped. Patients with class V caries usually consume large amounts of sugary carbonated drinks, sip on such drinks for several hours during the day, or produce low amounts of saliva. Drugs and therapies that dry the mouth are especially causative (see Figs. 83.1 and 83.2, **Meth Mouth**). Radiographically, class V caries should be distinguished from cervical burnout and toothbrush abrasion. In Figure 29.4, a typical class V lesion is present on the first premolar; the second premolar has recurrent decay beneath the amalgam restoration, and toothbrush abrasion is evident along the distal-cervical surface as a V-shaped radiolucent area. Smaller class V lesions can be treated with disking and fluoride varnish to remove either whitish or brownish stains without further treatment. Cavitated lesions require composite resins or amalgam. Esthetic situations can require the use of porcelain bonded-laminate veneers.

Class VI Caries (Fig. 29.5) **Class VI caries** is characterized by decay of the incisal edge or cusp tip. This type of caries is uncommon, but is more common in persons who frequently chew sugary gums or consume sticky candy bars. Patients with low salivary flow are also predisposed to this type of caries. Class VI caries—along with the class III caries and class I lingual pit caries—is seen in the shovel-shaped incisor syndrome.

Root Caries (Fig. 29.6) **Root caries**, also known as cemental caries, radicular caries, and senile caries, is decay that occurs on exposed root surfaces. It is detected

more often on posterior teeth in older patients. The elderly are more commonly affected because they have increased prevalence of (1) gingival recession; (2) periodontal disease, altered interproximal contacts, and food impaction; and (3) hyposalivation. Root caries starts at the cemento-enamel junction often on the interproximal surface below the contact point. However, it may be seen on any root surface. Early on, it appears as a shallow, ill-defined, softened and discolored area that tends to extend more circumferentially than in depth. Examination with an explorer reveals a soft rubbery consistency. As the lesion progresses, the decay surrounds the root, leading to the crown fracturing off; this is called **amputation caries** (Figs. 28.8 and Fig. 30.3). Progression is often fast because this region of the tooth has only a thin layer of enamel and dentin. Radiographically, root caries is seen in the interproximal areas below the cemento-enamel junction, and although neither the contact point nor the enamel is involved, the caries can spread coronally under the enamel and deep toward the pulp. Root caries often recurs when root surfaces remain exposed, salivary flow remains low, and oral hygiene does not improve. Frequent prophylaxis and home daily applications of fluoride are often needed. Restorations are complicated by the fact that margins end on cementum, and often there is little tooth structure remaining between the carious lesion and the pulp.

Recurrent Caries (Secondary Caries) (Figs. 29.7 and 29.8) **Recurrent caries** is defined as decay within the immediate vicinity of a restoration. It may be a sign of high caries risk, poor oral hygiene, or a defective restoration. Recurrent caries begins at a failing (ditched or leaking) margin of any restoration. These defective margins are predisposed to the accumulation of bacteria and food, and are protected from usual oral hygiene measures. Lesions progress at variable rates, appear dark, and are soft to the touch of the explorer tine. Radiographically, recurrent caries presents in two opposite ways. First, the decay may be seen as a radiolucent area beneath a restoration with or without a visibly defective margin, as seen in Figure 29.8 beneath the distal amalgam of the mandibular second premolar. Second, the decay may be seen as one or several flame-shaped or arrow-shaped radiopaque areas occurring uniquely beneath an amalgam-type restorative material, with the point of the arrow directed toward or encroaching on the pulp. In Figure 29.8, this phenomenon is seen beneath the mesial amalgam of the mandibular first molar. An associated radiolucent area is sometimes present as in this case. Spectroscopy studies have shown the radiopaque material represents softened dentin into which radiopaque zinc from the adjacent amalgam restoration has been leached. Recurrent caries are treated by removing the decay and replacing the restoration.



Fig. 29.1. Class IV caries: mesial of lateral incisor.



Fig. 29.2. Class IV caries: mesial of lateral; lingual caries.



Fig. 29.3. Class V caries: maxillary anteriors.



Fig. 29.4. Class V caries: first premolar.



Fig. 29.5. Class VI caries: premolar cusp tip.



Fig. 29.6. Root caries: on extruded molar.



Fig. 29.7. Recurrent caries: evident around restoration.



Fig. 29.8. Recurrent caries: lower premolar and molar.

DENTAL CARIES AND SEQUELAE

Caries Progression (Figs. 30.1–30.8) The invasion of caries may be a slow or fast process and may involve the pulp before the patient is aware of the lesion. In most cases, it takes several years for caries to reach the pulp. Some caries are particularly aggressive or have a unique cause. These caries have descriptive definitions. For example, **rampant caries** is a type of caries that develops at an extremely rapid pace in some children and young adults (see also “Meth Mouth,” Fig. 83.1). **Radiation** or **amputation caries** occurs and progresses rapidly in patients who have received radiotherapy who lack the protective action of saliva. This caries appears along the gingival margin of teeth and can weaken teeth so severely that the crown fractures. **Root caries** has an appearance similar to that of radiation caries but is not associated with a history of radiation therapy. Rather, these patients usually have a history of xerostomia. Root caries progresses more slowly than radiation caries because xerostomia is less severe. **Nursing bottle caries** results from prolonged contact of primary teeth with sugar-containing liquids in the baby bottle in infants.

If caries is left untreated, the bacterial infection can progress through tooth dentin and produce pulpal inflammation. The initial stage of pulpal involvement, **reversible pulpitis**, is characterized by pulpal hyperemia and tooth sensitivity to hot and cold that dissipates when the temperature source is removed. Persistent pulpal inflammation produces irreversible changes, or **irreversible pulpitis**, in which the patient experiences spontaneous and persistent pain in the tooth after removal of the temperature source. Severe destruction of pulpal tissue from bacterial infection or interruption of the blood supply to the pulp produces a **nonvital pulp** and subsequent periapical changes (chronic periapical inflammation).

Prevention is the best way to reduce the incidence and progression of caries. Clinical examinations are provided at least twice a year to minimize the sequelae of caries. Bitewing radiographs should be taken at 6-month intervals in children if clinical caries are detected or the patient has a high risk for caries. Adults at high risk should receive bitewing radiographs annually. Sealants should be placed over deep fissures of posterior teeth in caries-susceptible youths. Remineralization products, such as fluoride, fluoride varnishes, and certain sugarless gums, are advocated to prevent caries. Pulp vitality should be tested when a carious lesion has invaded to or beyond 50% of the space between the DEJ and pulp margin.

Pulp Polyp (Fig. 30.2) The **pulp polyp** is an inflamed pulp resulting from chronic bacterial infection in which a red, nonpainful, soft tissue mass grows out of the affected pulp. Extensively carious primary molars and 6-year molars of young children are most frequently affected. Although the tooth is initially vital, the condi-

tion eventually erodes and results in nonvitality. Treatment is extraction or root canal therapy.

Periapical Inflammation (Apical Periodontitis) (Figs. 30.4–30.6)

Periapical inflammation and **apical periodontitis** are clinical terms used to describe the radiographic changes and clinical findings associated with inflammation extending from the pulp chamber into the adjacent periodontal ligament around the apical foramen of a chronically inflamed tooth. The condition is most commonly associated with pulpal degeneration (nonvital tooth) but may occur in vital teeth from occlusal trauma or constant and repetitive pressure placed on a tooth. Radiographs show a widening of the apical periodontal ligament space. The **chronic** form may be symptomatic or asymptomatic. In contrast, **acute periapical inflammation** is painful. Both conditions cause the apical periodontal ligament to be sensitive to percussion. Chronic periapical inflammation has chronic inflammatory cells at the periapex and often shows greater periradicular destruction of alveolar bone than acute periapical inflammation.

Periapical tissue produces two usual responses to the degradation products of a nonvital pulp: either a periapical granuloma or periapical cyst. Both originate from a nonvital tooth and both have a similar radiographic appearance (round radiolucency at the apex of a nonvital tooth). The distinction is made histologically. The **periapical granuloma**, the most common response, consists of an accumulation of granulation tissue, lymphocytes, plasma cells, histiocytes, and polymorphonuclear leukocytes. The **periapical cyst** arises from a pre-existing periapical granuloma when inflammation stimulates proliferation of periapical epithelial rests of Malassez. Regression of the inflammation occurs after root canal therapy in most cases.

Periapical Abscess (Figs. 30.7 and 30.8)

A **periapical abscess** is the acute phase of an infection that spreads from a nonvital tooth through the alveolar bone into the adjacent soft tissue. An abscess is composed of neutrophils, macrophages, and necrotic debris. Clinical examination shows a red or reddish yellow, swollen nodule that is warm and fluctuant to the touch. The affected tooth is tender to percussion, slightly extruded, and responds abnormally or not at all to heat, cold, and electric pulp testing. Abscesses can be drained by opening into the pulp chamber or incising the soft tissue swelling. An alternate treatment is tooth extraction, which provides an avenue for drainage. Antibiotics are used when the abscess is large and spreading, lymphadenopathy and fever are present, and drainage is not established. Penicillin VK is the antibiotic of choice. If the infection is unresponsive to penicillin or rapidly spreading, a bacterial culture and sensitivity should be obtained. Antibiotics are generally not needed if the affected tooth is extracted, adequate drainage is established, and the patient is otherwise healthy.



Fig. 30.1. Extensive caries: mandibular molar with parulis.

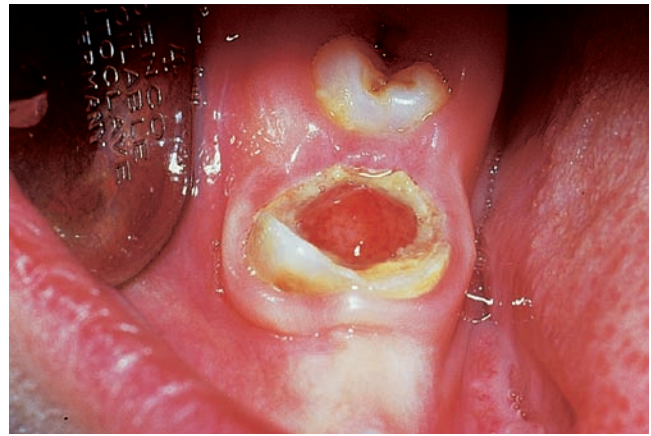


Fig. 30.2. Pulp polyp: red, exuberant mass arising from pulp.



Fig. 30.3. Rampant caries: associated with xerostomia.

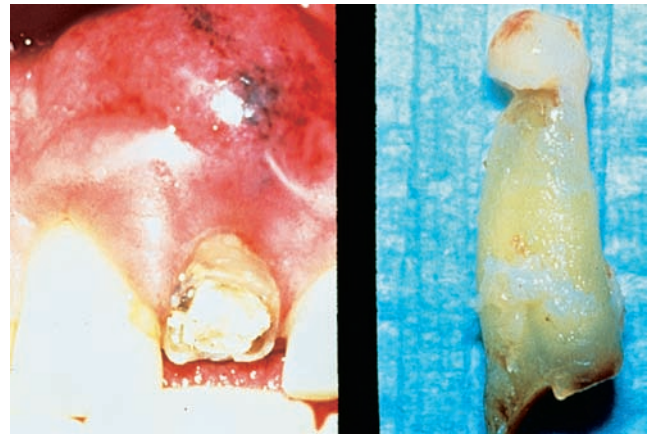


Fig. 30.4. Periapical inflammation: granuloma at apex.



Fig. 30.5. Periapical inflammation: draining parulis periapical no. 10.*



Fig. 30.6. Periapical inflammation.*



Fig. 30.7. Caries, chronic periapical inflammation, abscess first molar.†



Fig. 30.8. Abscess: draining through mandible.‡

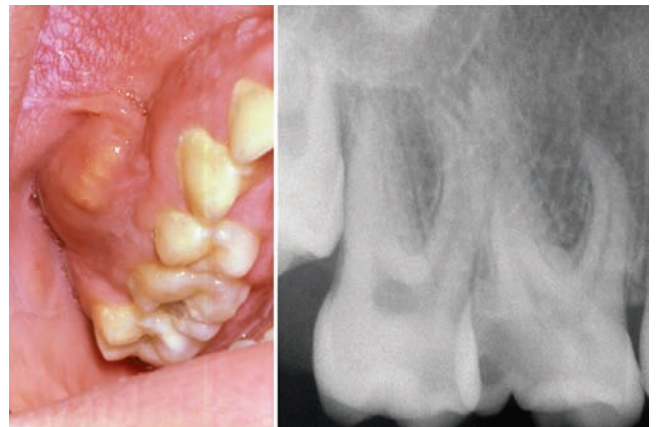
CASE STUDIES

CASE 9. (Fig. 30.9) This 14-year-old boy presents with this condition. He is unaware of the exact duration but remembers the tooth breaking off a few weeks ago. He has been chewing on the other side, but still little pieces seem to break off every few days. His mother wants to know what is going on, and can it be fixed?



1. Describe the most prominent findings in this quadrant.
2. Is this a normal finding, a variant of normal, or disease?
3. Are caries evident in this quadrant? If so, which teeth have caries?
4. What could you do to make the clinical assessment more effective?
5. Which of the following actions should the dental hygienist consider first?
 - A. Calculate his plaque score.
 - B. Conduct a gingival index.
 - C. Discuss with the dentist the need for x-rays.
 - D. Recommend fluoride varnish.
6. The most likely diagnosis for the condition affecting the mandibular first molar is:
 - A. Leukemic infiltrate
 - B. Pyogenic granuloma
 - C. Pulp polyp
 - D. Irritation fibroma
7. Is the pulp vital?
8. What other features are apparent on the teeth in this quadrant, and what are they suggestive of?

CASE 10. (Fig. 30.10) This young girl presents with the condition shown in the clinical and radiographic image. She says that the inside of her mouth in this region has gotten larger during the last 2 days.



1. How old is this girl?
2. Describe the clinical findings in this quadrant, as well as the radiographic findings.
3. Is this a normal finding, a variant of normal, or disease?
4. Do you expect this condition to be painful, and if so, why?
5. The most likely diagnosis for the condition affecting the primary maxillary second molar is:
 - A. Leukemic infiltrate
 - B. Lipoma
 - C. Pulp polyp
 - D. Periapical abscess
6. Is this condition limited to the primary teeth?
7. What are the treatment options for this tooth?

Radiolucent and Radiopaque Lesions of the Jaws

Dental Objectives:

- Define and identify common cysts and tumors of the jaws.
- Recognize the causes and clinical features of these conditions.
- Use the diagnostic process to distinguish similar-appearing anomalies of the jaws.
- Describe the consequences of disease progression with respect to jaw cysts and tumors.
- Recommend appropriate treatments for these anomalies.

Dental Hygiene Objectives:

- Recognize deviations from normal on dental radiographs.
- Describe the common radiographic appearance of cysts and tumors of the jaws.
- In the clinical setting, recognize deviations from normal on radiographs and call them to the attention of the dentist.
- Identify conditions discussed in this section that affect the delivery of periodontal debridement and oral hygiene measures.

RADIOLUCENT LESIONS OF THE JAWS: CYSTS

Cysts of the Jaws (Figs. 31.1–31.8) Cysts are abnormal epithelial-lined saclike (often fluid-filled) structures within tissue. These pathologic cavities develop commonly in jaws when epithelial remnants of developing or erupted teeth or embryonic structures undergo cystic degeneration. Jaw cysts tend to be asymptomatic and slow growing. They are classified as developmental, odontogenic, inflammatory, and pseudocysts (not epithelium lined), and appear as unilocular or multilocular radiolucencies. Multilocular types are aggressive and more likely to recur.

Nasopalatine Duct Cyst (Incisive Canal Cyst) (Fig. 31.1) This developmental cyst arises from entrapped epithelium in the incisive canal. Radiographically, it produces a classic heart-shaped radiolucency between the roots of vital maxillary central incisors (see “**Swellings of the Palate**,” Figs. 52.3 and 52.4). The **cyst of the incisive papilla** (Fig. 31.1) is a soft tissue variant of the incisive canal cyst. It occurs within the incisive papilla as a slow-growing, dome-shaped swelling. Occlusion against the lesion can cause it to appear red or ulcerated. Mastication-associated pain may be a symptom. Excision is the treatment of choice. Recurrence is rare.

Lateral Periodontal Cyst (Fig. 31.2) This is a developmental nonkeratinizing odontogenic cyst that develops lateral to a tooth root, commonly in the premolar-canine region. It arises from proliferation of epithelial rests of dental lamina and occurs mostly in the mandible in men between 40 and 70 years. It appears as a small (<5 mm), well-defined, corticated interdental radiolucency that contacts the mid- and coronal portion of the root. Adjacent teeth are vital. It has two variants: the **gingival cyst of the adult** and the **botryoid lateral periodontal cyst**. The gingival cyst is found entirely within soft tissue (Fig. 11.8). Gingival and lateral periodontal cysts rarely recur after excision.

Botryoid Lateral Periodontal Cyst (Fig. 31.3) This cyst is a multilocular variant of the lateral periodontal cyst. Unilocular variants occasionally occur. It appears as a cluster of grapes (botryoid). They occur slightly more often in men after age 40 years. The most frequent symptom is swelling or pain in the area. Most are found in the mandibular canine, premolar, and incisor areas. In this example, one of the locules is in the usual location of a simple lateral periodontal cyst, the remaining smaller locules are apically located. About 10 years after removal, 30% to 50% recur.

Dentigerous Cyst (Fig. 31.4) The dentigerous cyst associates with the crown of an erupting or impacted tooth. It arises from proliferation of remnants of the **reduced enamel epithelium** (sulcular epithelium). It is the most common pathologic pericoronal radiolucency and second most common jaw cyst after the periapical cyst. It is detected usually in persons between 10 and 30 years of age, more often in males. Radiographically, the cyst is a well-corticated pericoronal radiolucency of varying

size that is attached to the tooth at the cervical margin. It is histologically similar to the normal follicular sac, but produces a larger radiolucency than the normal follicular space; the latter is <2.5 mm in intraoral radiographs and 3.0 mm on panoramic radiographs. The most common location is the mandibular third molar region (56%). They can give rise to an **ameloblastoma**, **squamous cell carcinoma**, or **mucoepidermoid carcinoma**. Thus, excision is essential.

Odontogenic Keratocyst (OKC) (Figs. 31.5 and 31.6) This jaw cyst arises from remnants of the dental lamina and is defined by its histologic appearance. It frequently appears as a **primordial cyst** when a tooth (e.g., third molar) fails to develop. Most are asymptomatic, multilocular radiolucencies that occur in the molar region of young men (10 to 30 years of age). The margins are smooth, corticated, and often scalloped. Internally, some septa and a cloudy lumen (representing desquamated keratin) are seen. They have remarkable growth potential, and bony expansion tends to occur anteroposteriorly within the medullary bone. Histologically, the cystic epithelium is uniform, 8 to 10 cell layers thick, and **parakeratinized**. After excision, the recurrence rate is about 30%.

Nevoid Basal Cell Carcinoma Syndrome (Gorlin-Goltz Syndrome) (Fig. 31.6) This autosomal dominant syndrome is characterized by multiple jaw OKCs, basal cell nevi on the skin, skeletal anomalies (bifid and other rib anomalies, hypertelorism), and soft tissue anomalies (prominent finger pads and palmar pitting of the hands). OKCs have a high recurrence rate.

Paradental Cyst (Buccal Bifurcation Cyst) (Fig. 31.7) This cyst arises along the buccal or distal aspect of an erupting molar as a result of entrapped **sulcular epithelium** and bacteria that accumulate below buccal cervical enamel extensions of lower first, second, and third molars, in descending order of frequency. Most occur in young males before age 16 years. Radiographically, these well-circumscribed pericoronal radiolucencies develop buccally (or occasionally distally) to an involved molar and are bounded by a thin or thick dense cortical line. Treatment should address the periodontal defect at the bifurcation with enameloplasty and irrigation. Involved third molars are usually extracted.

Simple (Traumatic) Bone Cyst (Fig. 31.8) This lesion is an intra-bony cavitation that affects the jaws or long bones. It is a pseudocyst, as it lacks an epithelial lining. It is believed to arise after trauma, although a history of trauma is not required. About 60% of cases occur in males. The average age of detection is 18 years. Most are asymptomatic and occur in the mandible molar-premolar area. It appears as a radiolucency with a scalloping superior radiopaque margin extending between roots and a rounded inferior margin. Adjacent teeth are generally vital. The mesiodistal dimension is usually wider than the superior-inferior. The cyst regresses spontaneously or after curettage.



Fig. 31.1. Cyst of the incisive papilla: entirely in soft tissue.

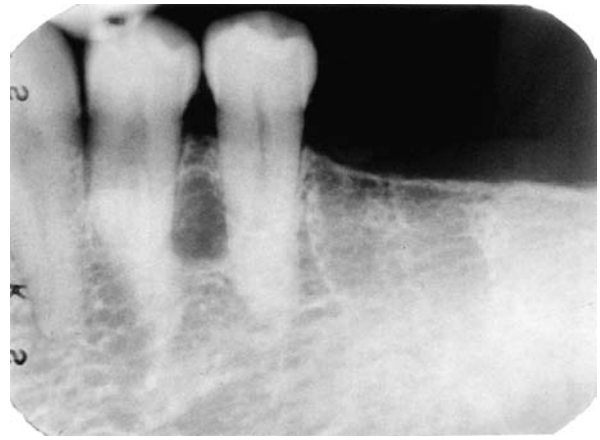


Fig. 31.2. Lateral periodontal cyst: common location and size.



Fig. 31.3. Botryoid lateral periodontal cyst: often multilocular.

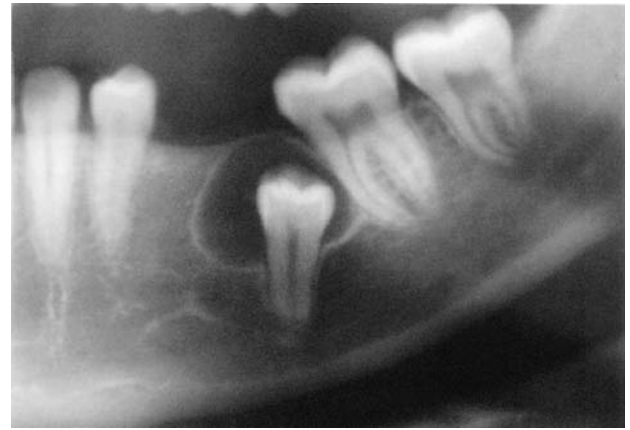


Fig. 31.4. Dentigerous cyst: in mandibular premolar location.

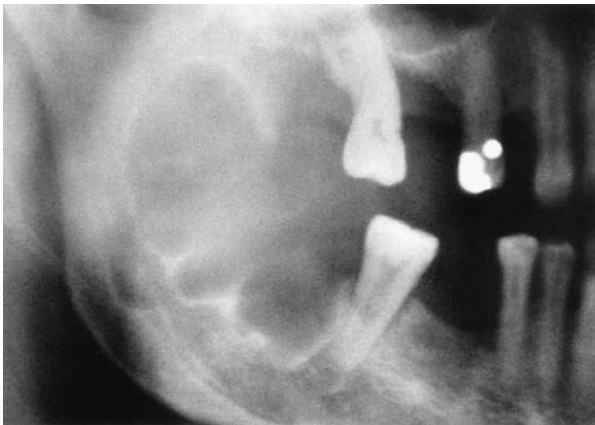


Fig. 31.5. Odontogenic keratocyst: multilocular and recurs.



Fig. 31.6. Nevus basal cell carcinoma: multiple OKCs.

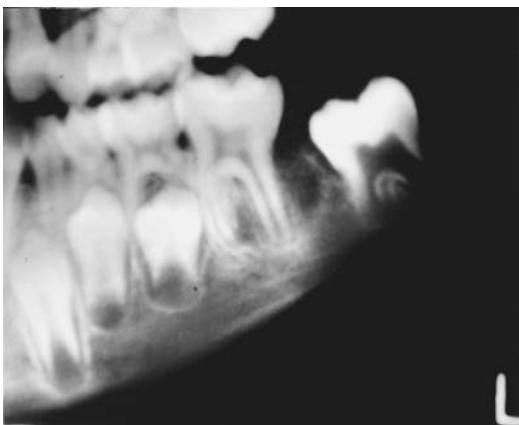


Fig. 31.7. Buccal bifurcation cyst: most common location surrounding the first molar.



Fig. 31.8. Traumatic bone cyst: oval and extending between roots.

RADIOLUCENT LESIONS OF THE JAWS: TUMORS

Unicystic (Mural) Ameloblastoma (Fig. 32.1) The **unicystic ameloblastoma** is an odontogenic tumor (an abnormal growth of neoplastic cells) that develops from **odontogenic epithelium** or from a pre-existing cyst. Most occur in the mandibular molar region of men, are painless swellings, and are diagnosed between age 20 and 30 years. Radiologically, this pericoronal radiolucency is often associated with a displaced third molar, buccal and lingual expansion, occasional perforation, and knife edge root resorption of adjacent erupted molars. Internally, there are locules or septa. They can perforate the cortical plate. Recurrence after treatment (enucleation and curettage) is infrequent.

Adenomatoid Odontogenic Tumor (Fig. 32.2) The **adenomatoid odontogenic tumor** is a slow-growing, mixed odontogenic neoplasm, derived from epithelial remnants of the enamel organ, that produces sheets of polyhedral epithelial cells and prominent ductlike structures. Most occur in young females (13 to 14 years); rarely, symptoms or swelling occurs. It has a striking tendency to produce a radiolucent lesion in the anterior jaw (maxillary canine area), which aids in the diagnosis. In about 65% of cases, radiopaque (enamel) flecks occur within the central part of the lesion. The margin of the tumor is radiopaque and may be thick, thin, or absent focally as a result of infection. An unerupted tooth (e.g., canine) is associated with these lesions in 75% of cases. Tumors contain a fibrous capsule, thus are easily enucleated, and tend not to recur.

Calcifying Epithelial Odontogenic (Pindborg) Tumor (Fig. 32.3) The **Pindborg tumor** is a painless, slow-growing swelling composed of sheets of large polyhedral neoplastic epithelial cells. The cause of the tumor is unknown. It occurs around age 40 years, equally in males and females, and, in two thirds of cases, in the mandibular molar area. Most are associated with an unerupted tooth and tumor-induced migration of a molar into the inferior cortex of the mandible. Smooth radiopaque flecks appear within the lesion, which cluster at the occlusal aspect of the tumor and form a linear “trail of driven snow” pattern. After excision, recurrence occurs in about 15% of patients.

Ameloblastic Fibro-Odontoma (Fig. 32.4) The **ameloblastic fibro-odontoma** is a mixed odontogenic tumor composed of neoplastic epithelium and mesenchyme. It occurs almost exclusively in tooth-development years (i.e., younger than 20 years) and more often in the posterior jaw. Males are affected slightly more often. The most frequent symptom is noneruption of one or several posterior teeth. It appears as a pericoronal radiolucency with internal radiopacities and hyperostotic margin, which often surround an unerupted tooth. The internal opacities contain variable amounts of calcified material (enamel and dentin) and may resemble a compound (toothlike) or complex odontoma. Expansion occurs with larger lesions. There is little tendency to recur after excision.

Odontoameloblastoma (Ameloblastic Odontoma) (Fig. 32.5) The **ameloblastic odontoma** is a rare variant ameloblastoma that demonstrates focal odontoma differentiation. Most occur in the first two decades of life, with an equal sex distribution. They appear as expanding pericoronal radiolucencies with diffuse radiopacities and often are associated with one or more impacted or unerupted teeth in regions anterior to the first molar. Lesions are prone to recur after treatment.

Ameloblastoma (Fig. 32.6) **Ameloblastoma**, an aggressive and locally invasive tumor arising from odontogenic epithelium, is the second most common odontogenic tumor. The average age at detection is about 34 years; men and women are equally affected. The tumor is characterized by slow growth and painless swelling that may reach huge proportions if untreated. The majority occur in the mandibular molar region, with some 60% extending into the ramus. The tumor is usually multilocular with bilocular, soap-bubble, and honeycomb variants; smaller lesions may be unilocular. Typically, both buccal and lingual expansion of the cortex is present in larger lesions, and perforation is possible. An impacted or displaced unerupted tooth is seen in about 40% of patients. Knife-edge resorption of adjacent teeth is characteristic. Ameloblastoma may metastasize; in this case, it is referred to as **malignant ameloblastoma**.

Myxoma (Fig. 32.7) The **myxoma** is a benign tumor that arises from odontogenic epithelial and mesenchymal (loose odontogenic pulp tissue) cells. Most occur in young adults (25 to 35 years), equally in men and women, and appear as a painless swelling associated with tooth displacement and cortical expansion of the posterior mandible. Maxillary lesions can involve the sinus and produce exophthalmos and nasal obstruction. Rare cases develop in the upper ramus and base of the condyle. Early lesions are unilocular, and advanced lesions are multilocular radiolucencies that have internal septa that intersect at right angles, forming geometric shapes. Perforation of the outer cortex and invasion of local soft tissue can produce a honeycomb appearance. About 30% recur after treatment.

Central Giant Cell Granuloma (Fig. 32.8) This inflammatory lesion is composed of multinucleated giant cells in a background of ovoid to spindle-shaped mesenchymal cells. More occur in women before age 30. There are two variants: aggressive and nonaggressive forms. The aggressive variant produces pain, a rapid-growing swelling (radiolucency exceeding 2 cm), radiographic resorption of root apices, and perforation of the expanded cortex. Nonaggressive lesions tend to be asymptomatic radiolucencies that exhibit slow growth and smaller size. Nearly 75% are seen in the mandible anterior to the first molars and cross the midline. The typical lesion is multilocular, with wispy trabeculae and crenations (scalloping) at the periphery. About 20% recur, especially the aggressive form.



Fig. 32.1. Unicystic ameloblastoma: cystlike with locules.

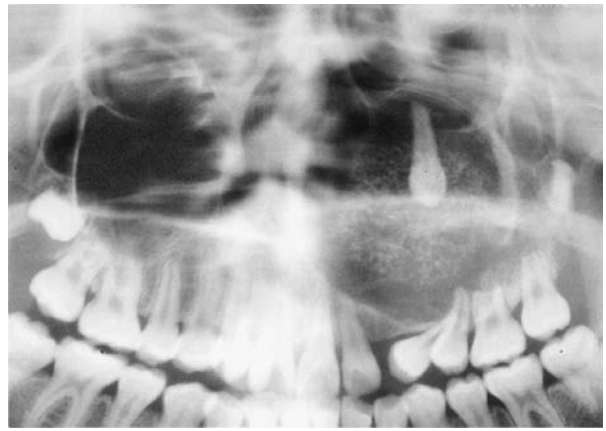


Fig. 32.2. Adenomatoid odontogenic tumor: with flecks.

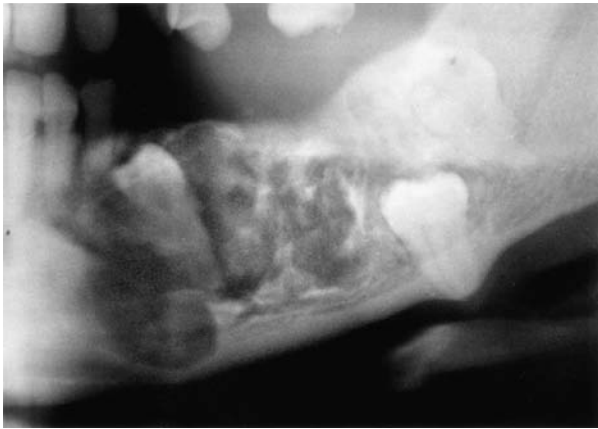


Fig. 32.3. Calcifying epithelial odontogenic (Pindborg) tumor.



Fig. 32.4. Ameloblastic fibro-odontoma: with nonerupted molar.



Fig. 32.5. Odontoameloblastoma: some locules with flecks.



Fig. 32.6. Ameloblastoma: expansile and soap-bubble locules.



Fig. 32.7. Myxoma: septa shaped like letters X, Y, and V.



Fig. 32.8. Central giant cell granuloma: peripheral crenations.

RADIOPAQUE LESIONS OF THE JAWS: BONY LESIONS

Exostosis (Figs. 33.1, 33.2, 69.5–69.7) **Exostoses** are asymptomatic, bony outgrowths of the outer cortex of the mandible and maxilla. Specific types include mandibular tori, palatal tori, and reactive subpontine exostosis. The clinical and histologic features are discussed under “**Nodules**.” All have a shell of cortical bone with various amounts of inner spongy (cancellous) bone. They occur on buccal or lingual alveolar bone as rounded bony nodules. They appear as dense, round radiopacities, thus they appear light on radiographs.

Mandibular Tori (Fig. 33.1) **Mandibular tori** are exostoses on the lingual alveolar bone adjacent to the premolars and canines, and sometimes molars. They are developmental outgrowths that occur in 10% of the population. Most show a hereditary pattern. Sizes range from 0.5 to 1.5 cm in diameter; however, they can slowly increase in size throughout life (see “**Nodules**,” Fig. 69.5). They appear as bilateral, focal, uniformly dense radiopacities with single or multiple lobes in the mandibular anterior and premolar periapical radiographs and occlusal and panoramic views. In periapical images, they are often superimposed on the roots of the teeth. The bosselated types appear as separate rounded or ovoid radiopacities with a smooth outline.

Palatal Tori (Fig. 33.2) **Palatal tori** are common bony hard masses that arise from the midline of the hard palate. These developmental and usually hereditary exostoses occur in 15% of the population, more often in women. Most appear as dome-shaped midline protuberances, but flat, nodular, spindle, and lobular variants exist (see Fig. 52.1). They consist of dense lamellar, cortical bone that can slowly increase in size. Typically, they are painless unless the overlying thin mucosa is traumatized. Radiographically, they appear as a focal, homogeneously dense radiopacity in the palatal region of maxillary anterior or posterior periapical radiographs and panoramic views. No treatment is required, unless desired for prosthetic reasons.

Osteoma (Figs. 33.3 and 33.4) An **osteoma** is a benign tumor composed of compact or cancellous bone. These bony hard masses arise in limited locations restricted to the craniofacial skeleton and rarely appear within soft tissue. Most appear in the posterior mandible or condyle as bony hard masses that arise from a polypoid or sessile base. When they extend beyond the confines of the parent bone, they are termed **peripheral osteoma**. Those confined within bone are known as **central osteoma**. The site of origin may be **periosteal** (lining of the cortex) or **endosteal** (from cancellous bone). They are generally painless and slow growing. Radiographically, they appear as a sclerotic mass of the same density as cortex. The borders are smooth, well defined, and rounded. Histologically, they appear identical to tori and exostoses. Peripheral osteomas are often removed for cosmetic and/or prosthetic reasons. Central osteomas usually are not removed unless there is extensive asymmetry

or interference with function. Multiple osteomas may be a sign of **Gardner syndrome** (Figs. 20.7 and 20.8).

Reactive Subpontine (Hyperostosis) Exostosis (Fig. 33.5) **Subpontic hyperostosis** is a reaction of crestal alveolar bone to the presence and occlusion of a mandibular fixed partial denture pontic. They occur in men twice as often as women, most after 40 years of age and most in the mandibular molar premolar region within 10 years of prosthesis placement. They have mild growth potential and can enlarge beneath the pontic, causing the tissue to become red or inflamed, or even to dislodge the bridge. Radiographically, they appear as a conelike radiopacity or broad-based opacity that completely fills the edentulous space. The radiographic margin is smooth and well defined. Regression occurs when the bridge is removed.

Socket Sclerosis (Fig. 33.6) **Socket sclerosis** is an asymptomatic, reactive lesion that occurs with systemic disorders (gastrointestinal [malabsorption] and kidney disease) after tooth extraction. It is a permanent marker of systemic disease even after the problem resolves. Most cases occur in the mandible after 40 years of age. There is no sex or racial predilection. Radiographically, it is characterized by (1) lack of resorption of the lamina dura, which usually resorbs 6 to 16 weeks after tooth extraction, and (2) deposition of sclerotic bone within the confines of the socket. Multiple root sites may be affected. This condition requires no treatment.

ROOT AND PERIAPICAL RADIOPAQUITIES

Idiopathic Osteosclerosis (Enostosis) (Fig. 33.7) **Idiopathic osteosclerosis** is bone deposition within marrow spaces of the jaws. There is no racial or sex predilection and no inflammatory or infectious cause. Most lesions are in the mandible in the premolar molar area. They appear as radiopaque densities or foci (55% of cases) at or near the apex of a tooth, in interradicular areas (28%), and distant to teeth (17%). When associated with teeth, the teeth are always vital (confirmed by vitality testing), and the apical periodontal membrane space is normal or rarely obliterated by the opaque bone. No treatment is required.

Condensing Osteitis (Fig. 33.8) **Condensing osteitis** is a proliferative reaction of dense bone deposited within marrow spaces of the jaws in response to pulpitis or pulpal necrosis. It is more common in persons younger than 20 years. Affected teeth are usually asymptomatic, but are dying. Most lesions are in the mandibular molar region. The lesion consists of a densely radiopaque area (sclerotic bone) localized to the apex of a tooth root, with widening of the apical periodontal membrane space. Often there is carious involvement of the pulp, or a large restoration or crown. Although the radiographic appearance is similar to periapical idiopathic osteosclerosis, the associated tooth is nonvital. Root canal therapy or extraction is recommended for the nonvital tooth. About 85% of cases regress, either partially or fully after therapy.

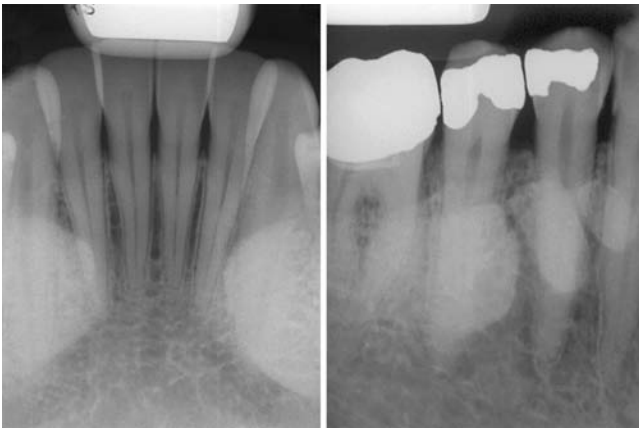


Fig. 33.1. Mandibular tori: typical locations.

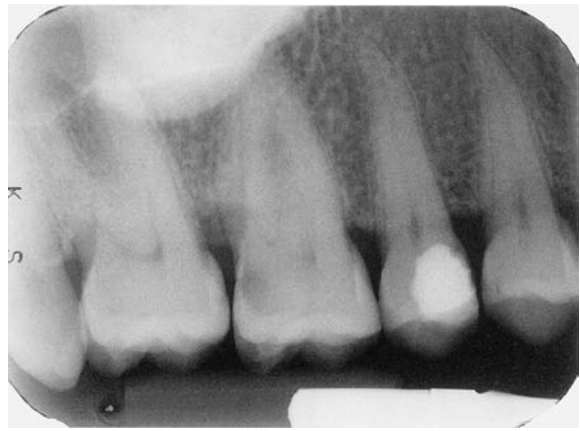


Fig. 33.2. Palatal torus: large rounded radiopacity.

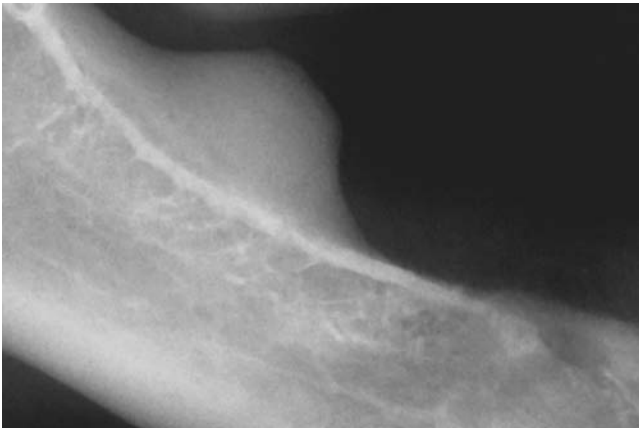


Fig. 33.3. Osteoma: posterior mandible; dense throughout.

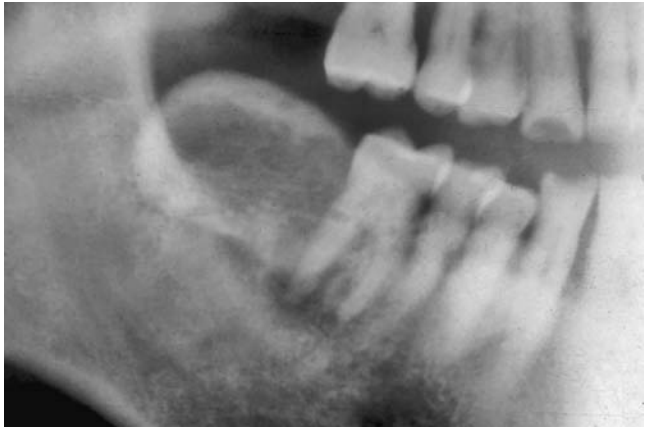


Fig. 33.4. Osteoma: posterior mandible; denser peripherally.



Fig. 33.5. Reactive subpontine exostosis: under pontic.



Fig. 33.6. Socket sclerosis: molar area; bulbous root premolar.



Fig. 33.7. Idiopathic osteosclerosis: vital second molar.



Fig. 33.8. Condensing osteitis: nonvital first molar.

RADIOLUCENT-RADIOPAQUE LESIONS OF THE JAWS

Odontoma (Figs. 34.1 and 34.2) The **odontoma**, although usually classified as an odontogenic tumor, is not a true neoplasm. It is considered a developmental anomaly (hamartoma) of enamel and dentin that exhibits defective architecture. Two forms exist: **compound odontomas**, the components of which assemble to resemble teeth, and **complex odontomas**, amorphous radiopaque masses that do not resemble teeth. Most are identified in teenagers and young adults on routine radiographs or when a permanent tooth fails to erupt. They are usually asymptomatic and single, but multiple odontomas are possible. Most occur in the anterior maxilla, with about two thirds occurring in the anterior jaws.

In the developing stages, radiolucent and mixed radiopaque components are seen, though the lesions are predominantly radiopaque. All are surrounded by a thin radiolucent zone corresponding to the follicular space of a normal tooth and an outer, thin corticated line. Odontomas have little growth potential unless they give rise to a dentigerous cyst. Removal is curative.

Compound Odontoma (Fig. 34.1) The **compound odontoma** is a mass resembling a conglomeration of rudimentary little teeth. They are twice as common in the maxilla as the mandible and occur in the anterior maxilla and with an unerupted tooth in 65% of cases. The odontoma is most often located next to an impacted tooth. Less commonly, the odontoma arises adjacent to or between tooth roots. In about half of cases, the crown or root of an adjacent tooth is impinged on by the odontoma, which can delay or alter its eruption (Fig 34.1B).

Complex Odontoma (Fig. 34.2) **Complex odontomas** are gnarled densities of enamel, dentin, and pulp that appear as solid radiopaque masses within bone. Unlike the compound odontomas, the majority of complex odontomas are found in the posterior regions of the mandible. They are associated with an impacted tooth in 70% of the cases, often above the impacted tooth, thus affecting its eruption.

Cemento-Osseous Dysplasias **Cemento-osseous dysplasias** are a group of reactive fibro-osseous lesions (discussed below) that progress through three radiographic stages: an early osteoporotic phase (radiolucent), followed by a mixed radiolucent/radiopaque lesion, and finally a radiopaque lesion surrounded by a thin radiolucent rim. The presence of a radiolucent rim helps distinguish these lesions from an osteoma and fibrous dysplasia.

Periapical Cemento-Osseous Dysplasia (Periapical Cemental Dysplasia, Cementoma) (Figs. 34.3 and 34.4) **Periapical cemento-osseous dysplasia** is an asymptomatic fibro-osseous disorder classified as a type of cemento-osseous dysplasia. It is seen almost uniquely in middle-aged women of African origin. The lesions are mixed radiolucent/radiopaque, occur usually in multiples, and are associated with the apices of the mandibular anterior teeth, which are vital. Three distinct stages occur. In stage I, the alveolar bone demonstrates periapical osteoporotic change. Stage II

(cementoblastic stage) demonstrates a frank radiolucency resembling a periapical radiolucency. In stage III (mature stage), calcific spherules develop within the radiolucencies that coalesce to form a central radiopaque mass. Stage III lesions have a “target” appearance. After pulp testing, no treatment is required.

Focal Cemento-Osseous Dysplasia (Fig. 34.5) **Focal cemento-osseous dysplasia** is a reactive lesion, similar to periapical and florid cemento-osseous dysplasia, except this entity presents as a single lesion in the posterior mandible. Middle-aged white and black women are affected. The apical region of an existing vital posterior tooth (or extraction site) is the most common location. Lesions tend to grow inferiorly and laterally but are asymptomatic. Early lesions are radiolucent or mixed radiolucent/radiopaque. Mature lesions are targetlike with a central radiopacity, a surrounding radiolucency, and a thick or thin peripheral radiopaque margin. Lesions can be dormant or active. Treatment is not required.

Florid Cemento-Osseous Dysplasia (Fig. 34.6) **Florid cemento-osseous dysplasia** is a diffuse variant of periapical cemental dysplasia that demonstrates multiple involvement of the apices of the posterior teeth in bilateral quadrants. It is found predominantly in middle-aged and older black women. Lesions progress through three stages (as described above), with the production of large cemental masses that coalesce. Lesions remain and continue to grow and coalesce after tooth extractions. Focal mild expansion is occasionally described. Complications include the presence of simple bone cysts, infection, and discomfort. Trauma can induce ulcers and bony sequestration. Surgical intervention and antibiotics may speed healing.

Cementoblastoma (Fig. 34.7) The **cementoblastoma** is a slow-growing neoplasm of cementum seen usually between 10 and 30 years of age. It presents as a well-circumscribed and corticated radiolucency that contains a circular radiolucency surrounding a central radiopaque mass fused to the root of a tooth, usually a vital permanent mandibular first molar or lower premolar. The lesion markedly attaches itself to the root apex, is expansile, and causes pain. Treatment involves surgical extraction of the tooth and attached lesion or excision of the root and lesion followed by endodontic therapy.

Ossifying Fibroma (Fig. 34.8) The **ossifying fibroma** is a neoplasm of bone that has three histologic variants: ossifying, cemento-ossifying, and cementifying. The tumor affects the posterior mandible in about 90% of cases and produces one of three radiographic patterns: a radiolucency that is well demarcated or corticated to a mixed radiolucent/radiopaque mass with a peripheral radiolucent rim. It is typically slow growing, round, and expansile with characteristic displacement of teeth and downward expansion of the inferior border of the mandible. Most often, middle-aged white women are affected. A juvenile form exists.

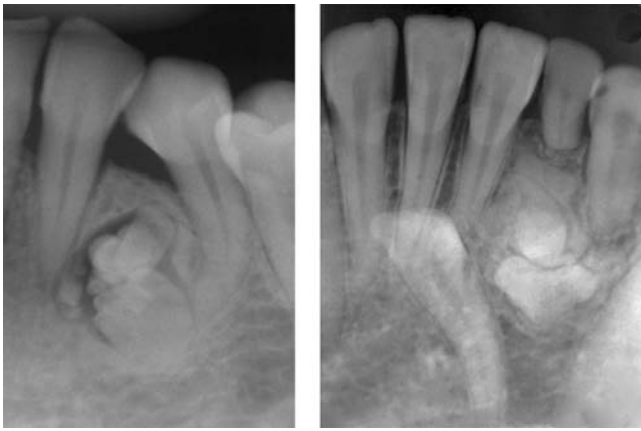


Fig. 34.1. Compound odontoma: affecting adjacent teeth.

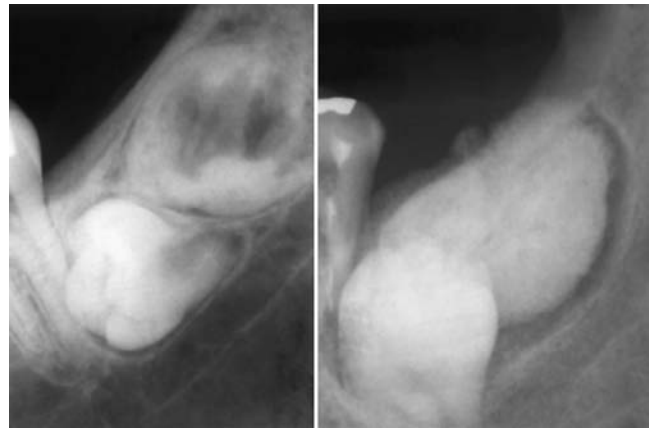


Fig 34.2. Complex odontoma: related to impacted teeth.



Fig. 34.3. Periapical cemento-osseous dysplasia: multiple stages.



Fig. 34.4. Periapical cemento-osseous dysplasia: stage III.



Fig. 34.5. Focal cemento-osseous dysplasia: single and posterior location.

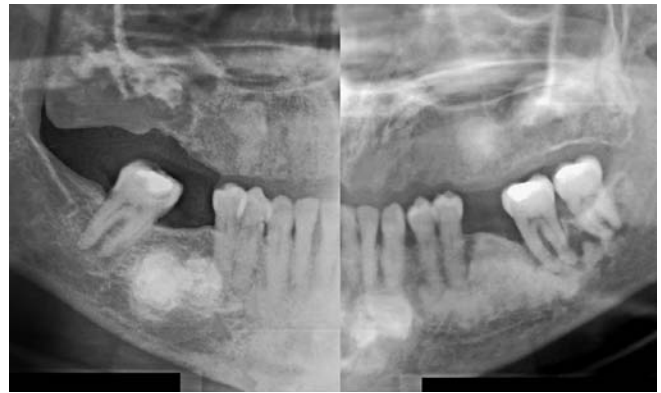


Fig. 34.6. Florid cemento-osseous dysplasia: all four quadrants affected.



Fig. 34.7. Cementoblastoma: fused to and obscuring the root apex.



Fig. 34.8. Ossifying fibroma: early developing lesion in a 37-year-old.

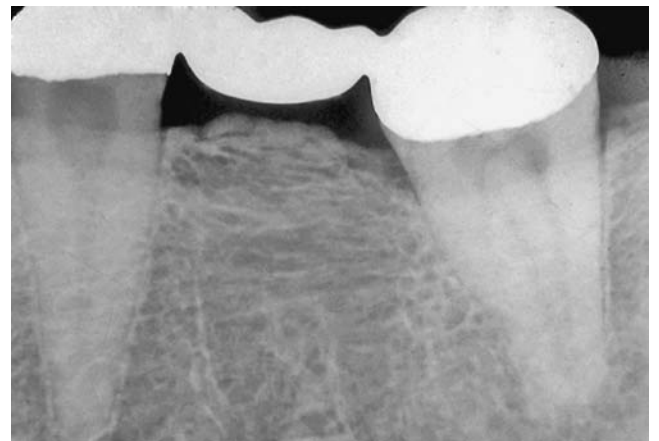
CASE STUDIES

CASE 11. (Fig. 34.9) This 43-year-old African American woman came to the dental clinic because she wants her teeth cleaned and is interested in getting a crown on tooth no. 27. She had a root canal completed on tooth no. 27 (right mandibular canine) a few months ago by an endodontist in another city and just moved to your town. She is a good dental patient who goes to the dentist every 6 months for cleanings.



1. Describe the radiographic findings.
2. Which term best describes the lesion at the apex of no. 25?
 - A. Radiopacity
 - B. Radiolucency
 - C. Radiodensity
 - D. Mixed radiopacity-radiolucency
3. Is this a normal finding, a variant of normal, or disease?
4. Do you expect tooth no. 25 to be vital or nonvital? Why? And how can you determine the vitality of tooth no. 25?
5. Do you expect this condition to be symptomatic?
6. List conditions you should consider in the differential diagnosis, and note which condition is most likely the correct diagnosis.
7. What precautions should be taken during periodontal debridement procedures? For example, is tooth no. 25 likely to be mobile?
8. Why do you think a root canal was performed on tooth no. 27?

CASE 12. (Fig. 34.10) You take this radiograph of a 55-year-old man in your dental office. He had the bridge constructed at another dental office and has no dental symptoms. He is hypertensive and takes a diuretic.



1. Describe the radiographic findings. Include normal and abnormal findings.
2. Do you expect this condition to be symptomatic? Why?
3. Is this a normal finding, a variant of normal, disorder, or disease?
4. Which of the following is the most likely diagnosis?
 - A. Mandibular enostosis
 - B. Reactive subpontine exostosis
 - C. Irreversible pulpitis
 - D. Condensing osteitis
5. This condition is more common in:
 - A. The maxilla of an older men
 - B. The mandible of an older men
 - C. The maxilla of a younger men
 - D. The ramus of an older men
6. What periodontal findings are associated with this condition?
7. If the bridge is removed and reconstructed, would the lesion regress?

Disorders of Gingiva and Periodontium

Dental Objectives:

- Define and use diagnostic terms that describe types of gingivitis, periodontitis, and diseases of the periodontium.
- Recognize the causes and clinical features of the different types of gingivitis, periodontitis, and diseases of the periodontium.
- Use the diagnostic process to distinguish the different types of gingivitis, periodontitis and diseases of the periodontium.
- Recommend appropriate treatment options for the different types of gingivitis, periodontitis, and diseases of the periodontium.

Dental Hygiene Objectives:

- Define the term *bacterial plaque*.
- Define, compare, and contrast the terms *gingivitis* and *periodontitis*.
- Define, compare, and contrast the terms *dehiscence* and *fenestration*.
- Define the term *periodontal abscess*.
- Define the terms *granuloma* and *fibroma*.
- Recognize clinically and radiographically abnormalities of the gingiva and periodontium.
- Identify conditions discussed in this section that (1) require the attention of the dentist and/or (2) affect the delivery of periodontal debridement and oral hygiene measures.

In the figure legends, * denotes the same patient.

PERIODONTAL DISEASES: PLAQUE, CALCULUS, AND REGRESSIVE CHANGES

Plaque (Figs. 35.1 and 35.2) **Plaque** is a biofilm of bacteria that adheres tenaciously to tooth surfaces, restorations, and prosthetic appliances. The development of plaque is a well-characterized, stepwise process. The first step is the attachment of the **acquired pellicle**, a thin film of salivary proteins. Within a few days, gram-positive facultative cocci overlay and colonize the pellicle, then additional bacteria, such as *Veillonella* species (a gram-negative anaerobe), *Actinomyces* species (a gram-positive rod), and *Capnocytophaga* species (a gram-negative rod) enter the region and colonize plaque. *Prevotella intermedia* and filamentous *Fusobacterium* species colonize between the first and third weeks as a subgingival **anaerobic** environment becomes established. Undisturbed plaque is colonized with *Porphyromonas gingivalis*, motile rods, and *Treponema* species (spirochetes) during and after the third week. The exact microbial composition varies according to site, available substrate, salivary components (adhesins and secretory immunoglobulin), duration, and the patient's oral hygiene practices.

Plaque is soft, translucent to white, and contains an extracellular, sticky matrix called **glucan**. Glucans are secreted by streptococci and promote adherence of bacteria to the pellicle. Clinically, plaque is classified by location as **supragingival plaque**, adhering to tooth structure above the gingiva, and **subgingival plaque**, found below the gingiva. Growth in supragingival plaque mass results from nutrients obtained from ingested simple carbohydrates (glucose) and lactic acid. In contrast, subgingival (plaque) bacteria preferentially use metabolized peptides and amino acids that are obtained from tissue breakdown products, the gingival crevicular fluid, and interbacterial feeding. Inflamed gingival tissues produce more gingival crevicular fluid, which favors the proliferation of subgingival bacterial replication. Subgingival bacteria prefer an anaerobic environment, whereas supragingival bacterial populations prefer a low-oxygen environment. The latter are called **facultative anaerobes**. Long-standing plaque is mostly composed of gram-negative anaerobes. Persistent microbial plaque can lead to stain, caries, gingivitis, calculus formation, gingival recession, and periodontitis. Poor esthetics, halitosis, and bacterial sepsis may be accompanying problems.

Calculus (Figs. 35.3–35.5) **Calculus** consists primarily of mineralized, dead bacteria with a small amount of mineralized salivary proteins. Its chemical components are mostly calcium phosphate, calcium carbonate, and magnesium phosphate. Calculus is hard, mineralized, and firmly adheres to the tooth. Above the gingival margin, calculus is called **supragingival calculus**. It appears yellow or tan and is usually located near large salivary sources in patients who do not mechanically remove plaque regularly. Supragingival calculus accumulates preferentially along the lingual of the mandibular

incisors adjacent to the duct for the sublingual and submandibular glands, and along the buccal of maxillary molars adjacent to Stensen's duct of the parotid gland. With age, it darkens and increases in size. A **calculus bridge** is an extensive matrix of calculus that extends across several teeth. It is often associated with gingival recession and periodontal disease. Removal of a calculus bridge may reveal several mobile teeth. Patients should be advised of this possibility before debridement.

Subgingival calculus forms below the gingival collar. It is not usually visible unless gingival recession has occurred. Subgingival calculus is detected with an explorer as a rough mass projecting from the cementum. It appears brown, black, or green because of its chronic exposure to gingival crevicular fluid, blood, and blood breakdown products. It is associated with the development of a **pyogenic granuloma** (Fig. 40.1), an epulislike lesion on the gingiva.

Gingival Recession (Figs. 35.6 and 35.7) In health, the gingival margin normally extends about 1 mm above the cemento-enamel junction. **Gingival recession** is the migration of the free gingival margin to a position apical to the cemento-enamel junction. Gingival recession is an indication of the apical migration of the junctional epithelium in the presence of disease and/or trauma. By definition, recession results in loss of attachment and exposure of cementum. It usually occurs in persons older than age 30 on the facial aspect of teeth. Recession may be localized or generalized and often progresses during periods of inflammation that may be combined with improper toothbrushing. Prominently located teeth with thin gingiva, inadequate bands of attached gingiva, high muscle or frenum attachments, bony fenestrations, and dehiscences are commonly affected. Excessive occlusal loading, temporary crowns, plaque, and calculus are also contributing factors. A variant form is **clefing**—a limited zone of narrow recession. Clefting can be caused by fingernail injuries, oral jewelry, or calculus. Surgical grafting is one treatment option that can provide root coverage for areas of clefting or recession.

Dehiscence and Fenestration (Fig. 35.8) A **dehiscence** is loss of alveolar bone on the facial—rarely lingual—aspect of a tooth that leaves a characteristic oval, root-exposed defect apical to the cemento-enamel junction. The defect may be 1 or 2 mm long or extend the full length of the root. Three features of a dehiscence are gingival recession, alveolar bone loss, and root exposure.

A **fenestration** is a window of bone loss on the facial or lingual aspect of a tooth that places the exposed root surface directly in contact with gingiva or alveolar mucosa. It can be distinguished from dehiscence in that the fenestration is bordered by alveolar bone along its coronal aspect.



Fig. 35.1. Plaque: stained pink by disclosing solution.



Fig. 35.2. Plaque: along gingival margins.



Fig. 35.3. Calculus: at interproximal and gingival margins.

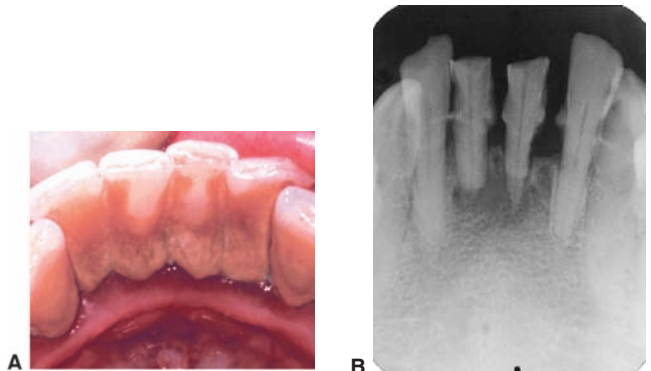


Fig. 35.4. A. Calculus bridge B. associated bone loss.



Fig. 35.5. Generalized gingival recession: 2 to 6 mm.



Fig. 35.6. Gingival recession: caused by frenal pull.



Fig. 35.7. Gingival clefting: beyond mucogingival junction.



Fig. 35.8. Dehiscences (D) and fenestrations (F).

GINGIVITIS

Gingivitis (Figs. 36.1–36.4) **Gingivitis** is a mixed bacterial infection that results in inflammation and reversible damage to the gingival tissues without loss of connective tissue attachment. It occurs at any age but most frequently arises during adolescence. It requires the presence and maturation of dental plaque. Gingivitis is diagnosed by bleeding and changes in the **color, contour** and **consistency** of the gingiva. Features include red swollen marginal gingiva, loss of stippling, red-purple, bulbous interdental papillae, and increased fluid flow from the gingival crevice. Bleeding and pain are induced by toothbrushing and slight probing.

Gingivitis has no sex or racial predilection and is classified according to distribution, duration, cause, and severity. The distribution may be general, local, marginal, or papillary (involvement of interdental papillae). The duration may be acute or chronic. Several different types are described in the literature and are discussed below. Treatment of gingivitis consists of frequent and regular removal of bacterial plaque. Untreated gingivitis can advance to periodontitis.

Gingivitis Caused by Mouth Breathing (Figs. 36.3 and 36.4) Chronic mouth breathing is characterized by nasal obstruction, a high narrow palatal vault, snoring, xerostomia, a sore throat upon wakening, and a characteristic form of gingivitis. Soft tissue changes are limited to the labial gingiva of the maxilla and, sometimes, the mandible. These changes may be an incidental finding or noticed together with caries limited to the incisors. Plaque accumulation at the gingival margin and multiple anterior restorations also serve as a diagnostic clue. Early changes consist of diffuse redness of the labial, marginal and interdental gingiva. The interproximal papillae become red, swollen and bleed. Progression results in inflammatory changes of the entire attached gingiva and bleeding on probing. Improved oral hygiene reduces these signs but does not resolve the condition. Protective dressings (i.e., emollients) placed on the affected gingiva promote healing. However, definitive treatment should address re-establishment of a patent (unobstructed) nasal airway.

Necrotizing Ulcerative Gingivitis (Fig. 36.5) **Necrotizing ulcerative gingivitis (NUG)** is a destructive infection, primarily of the interdental and marginal gingiva, characterized by partial loss of the interdental papillae, gingival bleeding and pain. The condition is also known as **Vincent's infection**, **pyorrhea**, or **trench mouth**, owing to its occurrence in men in battlefield trenches during the first World War. This multifactorial disease has a bacterial population high in fusiform bacillae, *Prevotella intermedia* and spirochetes. The condition is characterized by fever, lymphadenopathy, malaise, fiery red gingiva, extreme oral pain, hypersalivation, and an unmistakable fetor oris. The interdental papillae are punched out, ulcerated, and covered with a grayish pseudomembrane. The condition is common in persons between the

ages of 15 and 25 years, particularly students and military recruits enduring times of increased **stress** and reduced host resistance, and in HIV-infected patients. Smoking, poor nutrition, lack of sleep, and poor oral hygiene are contributory. In rare instances (such as malnutrition, malignancy, or immunodeficiency), the infection can extend to other oral mucosal surfaces, where it is known as **necrotizing ulcerative mucositis (NOMA)**. It can also recur if mismanaged. Treatment of NUG requires irrigation, gentle debridement, antibiotics (if constitutional symptoms are present), stress reduction, and rest. Partial loss of the interdental papillae can be expected despite normal healing.

Actinomycotic Gingivitis (Fig. 36.6) **Actinomycotic gingivitis** is a rare form of gingivitis that presents with redness, intense burning pain, and lack of a response to normal therapeutic regimens. Biopsy of tissue reveals the non-acid-fast, gram-positive, anaerobic bacteria (*Actinomyces*) forming filamentous colonies. Gingivectomy or long-term antimicrobial therapy provides effective treatment.

Focal Eruption Gingivitis (Fig. 36.7) **Focal eruption gingivitis** is a specific type of gingivitis seen around erupting teeth, usually canines or premolars. The condition, although not well documented in the literature, is rather common. It occurs as a hyperplastic reaction to microbial plaque around teeth that do not have adequate marginal and attached gingiva. It occurs most often in adolescents, whose dental arch lacks space for a late erupting tooth. Because the canines and premolars erupt late within the confined space, their eruption tends to be superior and facial. At this location, the collar of the tooth is surrounded by alveolar mucosa, which lacks the tensile and compressive properties of attached gingiva. The accumulation of plaque causes the tissue to become inflamed and fiery red. Inspection reveals minute red papules and a bandlike appearance around the collar of the tooth. The condition improves with meticulous oral hygiene and eruption into the normal position. In some cases, orthodontics may be required to position the tooth properly where attached gingiva can form.

Prophy Paste (Foreign-Body) Gingivitis (Fig. 36.8) **Prophy paste gingivitis** or **foreign-body gingivitis** is a rare form of gingivitis that occurs shortly after dental prophylaxis. It appears to be caused by the penetration of prophylaxis paste into the gingival tissue and the resulting inflammatory response to the foreign material. It may occur more often after air-powder abrasive system treatments, as these treatments can cause erosive changes and gingival impregnation of cleaning materials. Clinically, the affected tissue is focal or multifocal and appears red and friable. The well-demarcated red areas often are sore or burn. Treatment includes a tissue biopsy to confirm the diagnosis, topical steroids, careful debridement, and improved home care. If unresponsive, excision and a free gingival graft can be provided.



Fig. 36.1. Marginal gingivitis: bright red and swollen.



Fig. 36.2. Gingivitis: mild, caused by mouth breathing.



Fig. 36.3. Gingivitis: more severe, caused by mouth breathing.



Fig. 36.4. Chronic gingivitis: severe and plaque related.



Fig. 36.5. Acute necrotizing ulcerative gingivitis.



Fig. 36.6. Actinomycotic gingivitis: affecting marginal gingiva.



Fig. 36.7. Focal eruption gingivitis: fiery red above canines.



Fig. 36.8. Prophy paste gingivitis: 1 week after prophylaxis.

PERIODONTITIS

Periodontitis (Figs. 37.1–37.8) **Periodontitis** is inflammation of the periodontium caused by persistent microbial plaque. It is characterized by progressive loss of epithelial attachment and destruction of the periodontal ligament and alveolar bone. It is preceded by gingivitis and dental plaque that contains many anaerobic species. The most common form, **chronic periodontitis**, increases in prevalence with age and progress episodically. During exacerbations, there is apical migration of the epithelial attachment, increased periodontal pocket depth (>3 mm), increased gingival crevicular fluid, loss of alveolar bone, and loss of connective tissue attachment. Disease activity is assessed by monitoring these findings clinically, radiographically, and supplementally by analyzing the content of gingival crevicular fluid and saliva, which contain inflammatory mediators. Periodontitis usually results in mobility, shifting, and loss of teeth. Nonvitality and periodontal abscess are two less common outcomes.

Chronic periodontitis is divided into three types (mild, moderate, and advanced) based on severity and can be localized or generalized. The other categories of periodontitis include aggressive periodontitis (previously prepubertal periodontitis and juvenile periodontitis), periodontitis as a manifestation of systemic diseases, necrotizing periodontal diseases, abscesses of periodontium, and periodontitis associated with endodontic lesions. The predominant species associated with chronic periodontitis are *Actinomyces naeslundii*, *Tannerella forsythus*, *Campylobacter rectus*, *Eikenella corrodens*, *Eubacterium* species, *Fusobacterium nucleatum*, *Peptostreptococcus micros*, *Prevotella intermedia*, *Porphyromonas gingivalis*, *Selenomonas sputigena*, *Streptococcus intermedius*, and *Treponema* species (*T. denticola*). Certain species such as *Aggregatibacter (Actinobacillus) actinomycetemcomitans* are detected more often with specific types of periodontitis (i.e., aggressive periodontitis). Risk factors for periodontal disease include smoking, aging, and certain systemic diseases (diabetes mellitus, white blood cell disorders, and Ehlers-Danlos syndrome). Treatment involves the removal of plaque, calculus and diseased cementum by periodontal instrumentation. Antibiotics (tetracycline and metronidazole) are used with aggressive periodontitis. Periodontal surgery is recommended for unresponsive sites after periodontal instrumentation is complete and good patient self-care is in place.

Mild Periodontitis (Figs. 37.1 and 37.2) **Mild periodontitis** is characterized by minor breakdown of pocket epithelium, migration of neutrophils, increasing population of plasma cells, apical migration of junctional epithelium, minor destruction of the connective tissue attachment, and localized resorption of alveolar bone. This stage is defined by **1 to 2 mm of clinical attachment loss**, periodontal probe pocket depths of 4 to 5 mm, class I furcation involvement, and alveolar crestal bone loss of 2 mm or less. The **class I furcation** is limited destruction of bone between the superior aspect of the roots

and the tooth crown that is detectable by 1 mm entry of an explorer or probe. Alveolar bone loss is determined clinically by periodontal probing and supplemented by the use of vertical periapical bitewing radiographs or subtraction radiography.

Moderate Periodontitis (Figs. 37.3–37.5) **Moderate periodontitis** is the second stage of chronic periodontitis. Microscopic examination shows ulceration of the pocket epithelium, infiltrating populations of plasma cells and T cells, significant apical migration of junctional epithelium and destruction of the connective tissue attachment and alveolar bone. The condition is defined by **3 to 4 mm of clinical attachment loss**, periodontal pocket depths of 4 to 6 mm, alveolar bone loss that is 3 to 4 mm, gingival exudate and bleeding. Horizontal bone loss, vertical bone loss, osseous defects (moats, craters), mobile teeth and class II furcation involvement are additional radiographic and clinical features of the disease. The **class II furcation** is a 2 to 4 mm defect of cortical and alveolar bone located superiorly between the roots.

Advanced Periodontitis (Figs. 37.6 and 37.8) Advanced periodontitis is characterized microscopically by major destruction of the pocket epithelium, connective tissue attachment, and alveolar bone and large populations of plasma cells and T cells. Advanced periodontitis is defined by **at least 5 mm of clinical attachment loss**. Typically, periodontal pocket depths exceed 6 mm; alveolar bone loss is more than 4 mm; and gingival recession, significant tooth mobility, and **class III furcation** involvement (a through-and-through bony defect) are seen.

Periodontal Abscess (Figs. 37.7 and 37.8) A **periodontal abscess** is a collection of pus resulting from pathogenic bacteria that are occluded (trapped) in the periodontal pocket of a pre-existing periodontal lesion. The condition produces a rapidly progressing, red-purple fluctuant swelling that distends the attached gingiva. The gingival surface is typically smooth, with loss of stippling of the free marginal groove. Occasionally, the surface is necrotic or has pus emanating. Affected teeth usually have a deep pocket, subgingival calculus, an occluded entrance into the periodontal pocket, furcation involvement and are mobile. Patients often report well-localized, dull, and continuous pain, especially if the purulent exudate has no avenue for escape. The pain intensifies when pressure is applied to the tooth or the overlying soft tissue. Use of a periodontal probe may initially produce discomfort but is often therapeutic for a short time because it may drain the abscess. Fever, malaise, lymphadenopathy and an unpleasant taste may accompany the condition. The pulp of the affected tooth usually tests vital although the periodontal infection may spread to the pulp via the apex of a lateral canal. Treatment is directed toward removal of necrotic material, adequate drainage, localized periodontal therapy and improved plaque control measures.



Fig. 37.1. Mild periodontitis: loss of attachment evident.



Fig. 37.2. Mild periodontitis: loss of crestal alveolar bone.

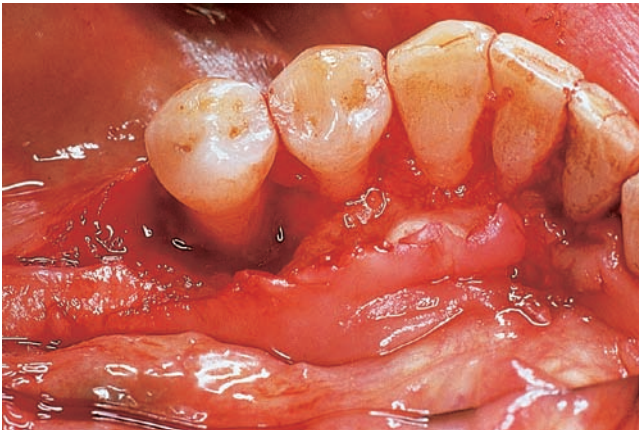


Fig. 37.3. Moderate periodontitis: 4-mm moat defect.



Fig. 37.4. Horizontal bone loss: and calculus.



Fig. 37.5. Class II furcation: a sign of moderate periodontitis.



Fig. 37.6. Class III furcation: advanced periodontitis.

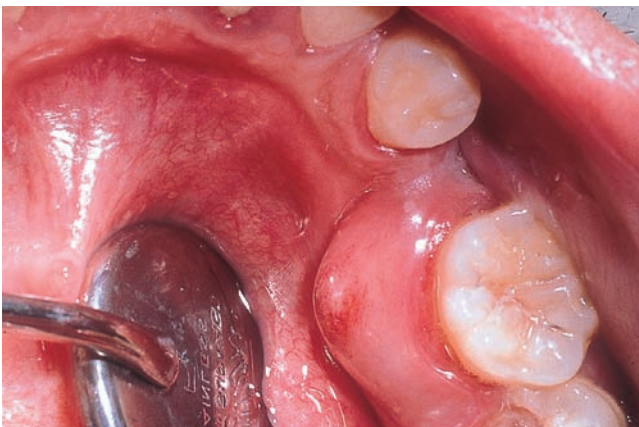


Fig. 37.7. Periodontal abscess: fluctuant and pointing.



Fig. 37.8. Periodontal abscess: with 12-mm pocket.

RADIOGRAPHIC FEATURES OF PERIODONTAL DISEASE

There are several radiographic features associated with periodontal disease, the most prominent being alveolar bone loss. This basic finding, although suggestive of periodontitis, is a nonspecific radiographic finding. It suggests that inflammatory and resorptive processes have been active. However, bone loss only provides historical reference. It is not indicative of whether the loss occurred years ago or recently.

Local Factors: Overhang (Fig. 38.1) Local factors can contribute indirectly to the development of periodontal disease. Examples include calculus (Fig. 37.4), crowding (Fig. 37.1), traumatic occlusion, supraeruption (Figs. 24.7 and 24.8), and restoration overhangs. An **overhang** is a specific portion of restoration that extends beyond the gingival preparation margin into the interproximal space. It is usually caused by inadequate banding and wedging of a prepared tooth before placement of the final restoration. Overhangs are seen with class II amalgams, as well as with composites and castings. Overhangs are **iatrogenic** because they are caused by a dental procedure. In Figure 38.1, a bone defect is associated with the mesial overhang of the maxillary second molar. There is also a small overhang on the mesial of the second premolar. However, it is associated only with a mild crestal irregularity. Overhangs should be removed and the margins smoothed either during periodontal procedures or as a new restoration.

Local Factors: Open Contact and Poor Restoration Contour (Fig. 38.2) Poor restoration contacts can contribute to food impaction, the accumulation of plaque, and the development of periodontitis. An **open contact** is an area between teeth where interproximal contact is absent. A poor restoration contact can be rough or have inappropriate width, length, height, or contour. Poor and open contacts can result from tooth mobility caused by malocclusion or periodontal disease, supraeruption, drifting of erupted teeth, attrition and flattening of the interproximal contacts, and iatrogenically (when a contact is improperly restored). In Figure 38.2, poor and open contacts are present between the first and second molars. Also, a bone defect is present, and the undercontoured restoration needs replacement.

Bone Loss: Localized (Figs. 38.1, 38.3, and 37.2) Healthy crestal alveolar bone is superiorly convex, densely radiopaque, continuous with the lamina dura on both sides, and located 1 to 1.5 mm apical to the cemento-enamel junction. Localized bone loss occurs when inflammatory cytokines cause bone resorption. Radiographically, one sees crestal irregularities, triangulation (bone defect formation), loss of bone density and interseptal bone changes. These changes may be localized or affect several areas. **Triangulation** is a wedge-shaped radiolucent defect that is a result of periodontal disease on the mesial or distal of the crestal interalveolar bone. Interseptal bone changes consistent with periodontal

disease are prominent nutrient canals, especially in the mandibular anterior region (Fig. 37.2 and 39.2). These canals appear as vertical radiolucent lines and indicate increased vascularity to the region and susceptibility to bone loss if treatment is not provided.

Bone Loss: Generalized (Figs. 38.4 and 37.4) Generalized bone loss indicates resorption of alveolar bone in multiple, often continuous sites. It occurs most frequently as **horizontal bone loss**. However, there may be areas of **vertical (or angular) bone loss**. In Figure 38.4, there is generalized horizontal bone loss, areas of vertical bone loss distal to the second premolar and mesial to the first molar, contact flattening caused by attrition, especially of the premolars, extrusion of the first molar with root caries, calculus, class III furcation involvement of the first molar, and furcation involvement of the second molar. The soft tissue outline of the gingiva seen along the cemento-enamel junction indicates that deep periodontal pockets are present.

Bone (Infrabony) Defects Angular or vertical bone defects are classified by the number of walls remaining after destruction of alveolar bone by periodontal disease. Although radiographs are suggestive of an infrabony defect and its severity, defects are best delineated by careful probing or surgical exploration. A **crater** is simply a scooped-out area of alveolar bone anywhere along the circumference of a tooth and is not classified as a walled defect.

One-Wall Infrabony Defect (Figs. 38.3 and 38.5) This defect has only one wall of interseptal bone (hemiseptum) remaining. The wall may slope mesiodistally, as seen distal to the second premolar in Figure 38.3, or ramp buccal or lingually, as seen in Figure 38.5 between the molars. This defect is difficult to treat.

Two-Wall Infrabony Defect (Fig. 38.6) A two-wall bony defect has two walls of interseptal bone remaining. Radiographically, there is a combination of the ramping, as seen in Figure 38.6, and the vertical defect, as seen in Figure 38.3. The result is a combination of bony walls creating an osseous defect in the interseptal bone at the mesial or distal of a tooth. This defect makes up 35% of all defects and 62% of mandibular defects. In our example, the crater is mesial alongside the lower second molar.

Three-Wall Infrabony Defect (Fig. 38.7) and Moat Defect (Fig. 38.8) This infrabony defect has bone on three sides with the tooth root forming the fourth wall. The defect surrounds only a portion of the root. When it involves the buccal or lingual surface or completely surrounds the root, it is referred to as a **circumferential** or **moat** defect. In the case in which the defect involves the periapex, inflammation and vascular compromise can result in necrosis of the pulp. This coalescence of infections is known as a combined periodontal-endodontic lesion.



Fig. 38.1. Local factors: overhang and crestal defects.



Fig. 38.2. Open contact, poor restoration contour: first molar.



Fig. 38.3. Vertical one wall defect: distal to premolar.



Fig. 38.4. Bone loss: generalized; extrusion and root caries.



Fig. 38.5. One-wall bone defect: ramping between molars.



Fig. 38.6. Two-wall cratering defect: around lower second molar.



Fig. 38.7. Three-wall bone defect: second molar area.



Fig. 38.8. Circumferential (moat) defect and nonvital tooth.

RADIOGRAPHIC ALTERATIONS OF PERIODONTAL LIGAMENT AND LAMINA DURA

Periodontal Ligament Space and Lamina Dura The **periodontal ligament (PDL) space** is a 1-mm radiolucent line surrounding the root (see example in Fig. 5.5). The **lamina dura** is a thin dense radiopaque layer of bone that lines the tooth socket. It is seen immediately outside the PDL space. Changes in the PDL space and lamina dura are caused by several diseases discussed below.

Acute Apical Periodontitis (Fig. 39.1) **Acute apical periodontitis** is a manifestation of pulpal inflammation and early necrosis that is due to deep caries, trauma, or operative procedures. The first sign is a localized thickening of the PDL space. Then osteoclasts resorb local bone, causing the lamina dura to become less distinct and ultimately fade. Subsequently, the apical PDL space widens, and resorption of apical alveolar bone occurs. Clinically, the tooth is tender to percussion. Without root canal treatment, a periapical abscess, cyst, or granuloma can be expected to form.

Periodontitis (Fig. 39.2) An early radiographic sign of **periodontitis** is disappearance of the lamina dura lateral to the root beginning at the alveolar crest. Resorption of the dura progresses apically. This initially produces a small triangular defect (**triangulation**) in the crestal bone with the apex of the triangle pointing apically. Another radiographic sign of periodontitis is the presence of prominent interdental nutrient canals (Fig. 39.2), especially in the mandibular anterior region. These canals are engorged blood vessels containing inflammatory elements that are prone to stimulate resorption and are risk factors for chronic active periodontitis.

Traumatic Occlusion and Orthodontic Tooth Movement (Fig. 39.3) **Traumatic occlusion** in one or more teeth can be seen radiographically. Typically, the tooth with a premature contact will have a prominently widened PDL space and a widened, more radiopaque lamina dura along one side of the involved tooth. Minor involvement limits the findings to the cervical half of the tooth. More severe cases involve the whole side of the tooth or both sides of the tooth. In this latter case, tooth mobility will be present. The PDL space can also be widened by **orthodontic treatment** (Fig. 24.2). Radiographically, one sees thinning of the PDL space and resorption of the lamina dura on the pressure side, and thickening of the PDL space and of the lamina dura on the tension side. When too much force is applied in a susceptible patient, external root resorption results (Fig. 27.1).

Scleroderma (Fig. 39.4) **Scleroderma** is a disease that slowly produces loss of elasticity in the skin and sclerosis as a result of deposition of collagen. It can be isolated or systemic. Patients develop difficulty in moving the fingers and in opening the mouth wide. In the jaws, the most prominent finding is a diffuse widening of the periodontal ligament space of multiple teeth in more than one quadrant. Bilateral resorption of the posterior border

of the ramus, coronoid process, and condyle can occur. When the condyles are resorbed, an anterior open bite will develop.

Malignant Disease-Related Periodontal Defect (Fig. 39.5) **Malignant disease** can mimic periodontal disease and involve the periodontium. In these cases, it causes a loss of the lamina dura and a thickened and irregular periodontal membrane space along the mesial or distal length of a tooth root. Sometimes several adjacent teeth are affected, or it can mimic a perio-endo lesion. The overlying gingiva may be red or enlarged, and the patient may be asymptomatic or report deep, debilitating bone pain. The most common malignancy to produce this is **metastatic disease**, especially in the mandible. Others include gingival carcinoma, osteosarcoma, and chondrosarcoma.

Ankylosis (Fig. 39.6) **Ankylosis** is defined as a fusion of the tooth root with alveolar bone. Many ankylosed teeth are retained primary molars (Fig. 19.6). Also affected are impacted permanent teeth and transplanted teeth, particularly mandibular third molars replacing first molars. Ankylosis is characterized by an absence of the PDL space and lamina dura, and a “submerged” crown below the normal plane of occlusion.

Fibrous Dysplasia (Fig. 39.7) **Fibrous dysplasia** is classified as a fibro-osseous disease whereby bone is replaced with a fibro-osseous connective tissue. Other findings are café au lait macules with borders resembling the coast of Maine. These patients are subject to pathologic fractures in affected weight-bearing bones and enlargement of the alveolar process and facial bones, which may create esthetic issues. The radiographic lesions can involve the jaws extensively with radiolucent, radiopaque, and mixed patterns. Along with these patterns, the lamina dura may be lost, and the adjacent bone may resemble ground glass. These changes are seen only in the bone affected by fibrous dysplasia.

Paget Disease and Hyperparathyroidism (Fig. 39.8) **Paget disease** (Fig. 39.8A) is a disease of irregular breakdown and formation of bone that result in bone expansion, weakening and pain. The cause is not known. It affects any bones, including the jaws, facial bones and skull. Affected bones symmetrically enlarge, requiring patients often to obtain a new hat and dentures. They also develop diastemas.

Hyperparathyroidism (Fig. 39.8B) is a disease of abnormal increase in circulating parathormone that is due to a parathyroid tumor, severe kidney disease, or both. Both Paget disease and hyperparathyroidism may demonstrate a generalized loss of the lamina dura and a ground-glass appearance of the alveolar bone. The PDL space is often thin but visible. In hyperparathyroidism, the osteoporotic bone is accompanied by the presence of brown tumors, which histologically resemble central giant cell granulomas.

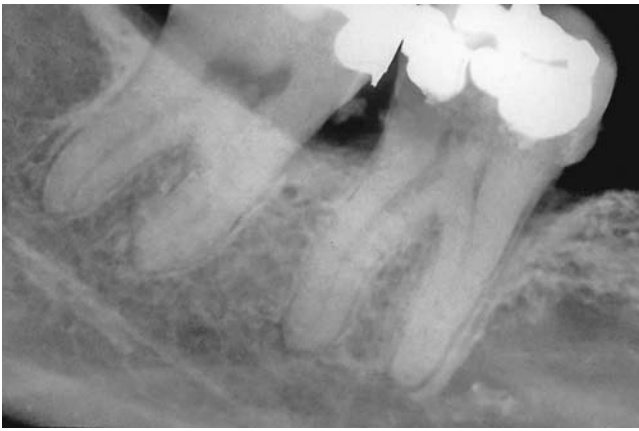


Fig 39.1. Acute apical periodontitis: lost lamina dura, thick PDL.

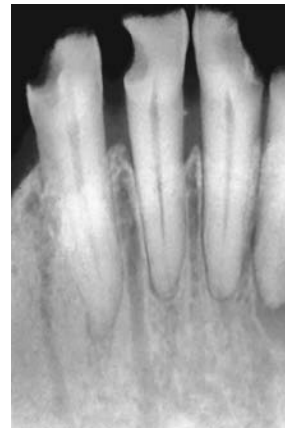


Fig. 39.2. Periodontitis: interradicular nutrient canals and funneling.



Fig. 39.3. Traumatic occlusion: no. 12 mesial wider lamina dura and PDL.

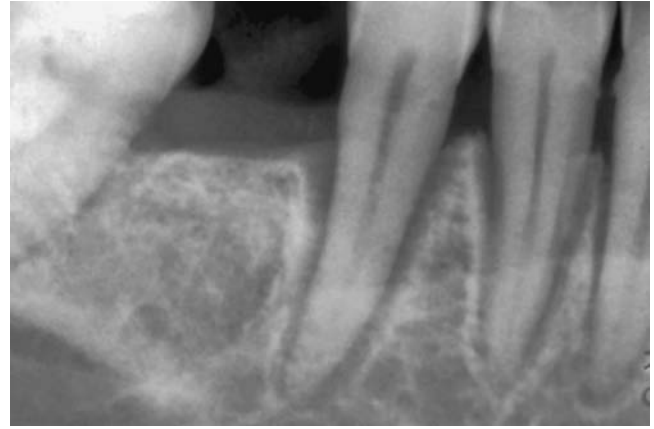


Fig. 39.4. Scleroderma: generalized widened PDL and lamina dura.

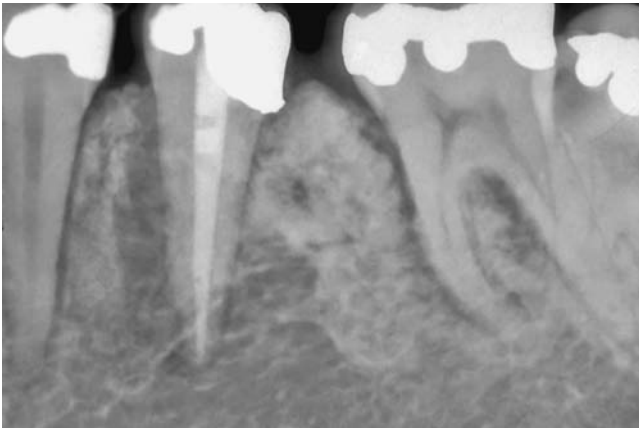


Fig 39.5. Malignant disease: chondrosarcoma; wide PDL one side root.

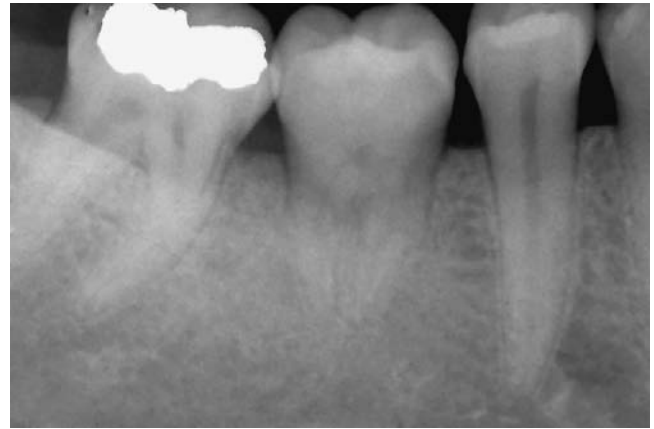


Fig 39.6. Ankylosis: transplanted third molar, no lamina dura or PDL.



Fig 39.7. Fibrous dysplasia: no PDL or lamina dura; granular bone.

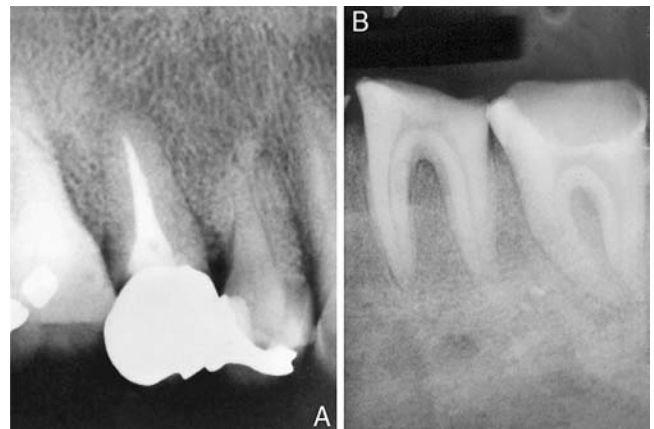


Fig. 39.8. A. Paget. B. Hyperparathyroidism: no PDL and lamina dura.

LOCALIZED GINGIVAL LESIONS

Pyogenic Granuloma (Figs. 40.1 and 40.2) **Pyogenic granuloma** is a common, benign mass of inflamed tissue that bleeds easily because of an abnormally high concentration of blood vessels. The name *pyogenic granuloma* is a misnomer because the condition is neither a granuloma nor pus filled. The growth is an exaggerated response to a chronic irritant, such as overhanging restorations or calculus. Often they develop rapidly and in patients who have poor oral hygiene. Women are more susceptible to the condition because of hormonal imbalances that occur during puberty, pregnancy, or menopause. In such cases, the granulomas are called **hormonal** or **pregnancy tumors**. About 1% of pregnant women develop this lesion.

Pyogenic granulomas appear as bright red, fleshy-soft nodules. The surface is glossy, ulcerated, and often lobulated. The base is polypoid or pedunculated. Although usually asymptomatic, the condition bleeds easily after minor manipulation because of the thinned epithelium and highly vascular tissue. Mature lesions become fibrotic, less vascular, and less red in color.

Pyogenic granulomas most frequently arise from the interdental papilla anterior to molar sites and can enlarge from the labial and lingual aspects to several centimeters. Other sites of development include the tongue, lips, buccal mucosa, and edentulous ridge. Treatment is surgical excision with removal of local irritants to prevent recurrence. In the pregnant woman, excision should be delayed until after childbirth.

Peripheral Giant Cell Granuloma (Figs. 40.3 and 40.4) The **peripheral giant cell granuloma** is a reactive lesion that develops exclusively on the gingiva. It is generally associated with a history of trauma or irritation and is thought to originate from the mucoperiosteum or periodontal ligament. Therefore, the peripheral giant cell granuloma demonstrates a restricted area of development—the dentulous or edentulous ridge. The mandibular gingiva anterior to the molars is particularly affected, especially in women between the ages of 40 and 60. Histologic examination shows multinucleated giant cells and numerous fibroblasts.

The peripheral giant cell granuloma is a well-defined, firm, epulislike growth that seldom ulcerates. The base is sessile, the surface is smooth or slightly granular, and the color is pink to dark red-bluish purple. The nodule is usually a few millimeters to 1 cm in diameter, although rapid enlargement may produce a large growth that encroaches on adjacent teeth. The lesion is generally asymptomatic. However, because of its aggressive nature, the underlying bone is often involved, producing a characteristic superficial “cupping” resorptive radiolucency of the alveolar bone. Treatment is excision that includes the base of the lesion and curettage of the underlying bone. Incomplete removal may result in recurrence. On histologic examination, this lesion cannot be distinguished

from the **central giant cell granuloma** and the **brown tumor of hyperparathyroidism**.

Peripheral Ossifying Fibroma (Figs. 40.5 and 40.6) The **peripheral ossifying fibroma** is a reactive growth, unrelated to the central ossifying fibroma, that is especially prone to occur in the anterior region of the maxilla of females in the second decade of life. The cause of the peripheral ossifying fibroma is uncertain, but inflammatory hyperplasia of superficial periodontal ligament origin has been suggested. The condition arises exclusively from the gingiva, usually the interdental papillae. The calcifications seen within the fibrous lesion may consist of bone, cementum, or dystrophic calcification. Usual clinical features of this solitary swelling are firmness, red or pink color, possible ulceration, and sessile attachment. An important diagnostic clue is the marked tendency of the condition to cause displacement of adjacent teeth. The chief sign is often an asymptomatic, slow-growing round or nodular swelling. Immature lesions are soft and bleed easily, whereas older lesions become firm and fibrotic. Radiographs may reveal central radiopaque foci, mild resorption of the crestal bone (peripheral cuff of bone), and wispy trabeculae. Treatment is excision. The recurrence rate is about 15%.

Irritation Fibroma The **irritation fibroma** is a common benign oral lesion that occasionally develops on gingival tissues. It is more typically seen on moveable mucosa and is discussed in Section 10 (Fig. 70.1).

Peripheral Odontogenic Fibroma (Fig. 40.7) The **peripheral odontogenic fibroma** is clinically similar to the irritation fibroma but characterized by its unique location and tissue of origin. In most cases, it presents as a firm, dome-shaped swelling on the facial aspect of the interdental papilla, generally located anterior to the molar teeth. A cuplike erosion of the underlying alveolar bone may be seen radiographically. The lesion probably arises from cellular components of the periodontal ligament. Microscopically, it shows strands of odontogenic epithelium among dense collagenous tissue and occasional mineralized products.

Desmoplastic Fibroma (Fig. 40.8) The **desmoplastic fibroma** is a rare tumor composed of dense fibroblasts and abundant collagen that most frequently affects the metaphyseal regions of the long bones of the arms and legs. The fourth most commonly affected site is the mandible. Most cases occur in adults younger than age 30. The posterior jaw is the most common intraoral location. The tumors begin as painless firm swellings within bone that expand and produce unilocular radiolucencies. Erosion of the cortical bone results in root resorption and a pink, firm soft tissue mass of the alveolar ridge or gingiva. The tumor is locally aggressive and recurs in about 30% of patients after surgical excision. Resection is recommended for recurrent lesions.



Fig. 40.1. Pyogenic granuloma: at interdental papilla.

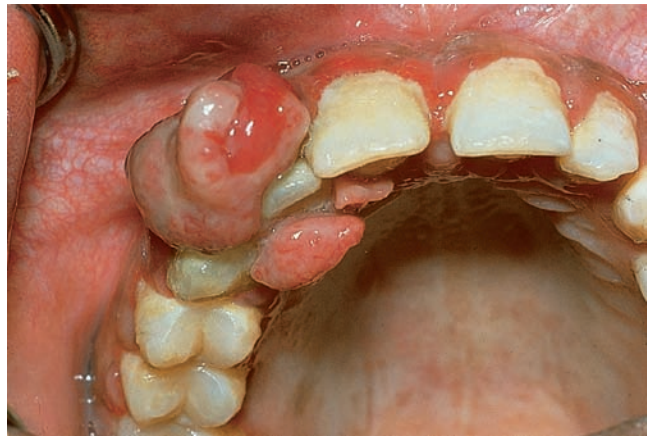


Fig. 40.2. Pregnancy tumor: 3 days after parturition.

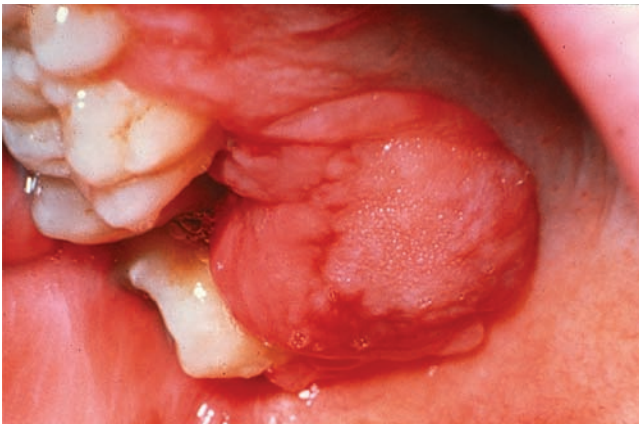


Fig. 40.3. Peripheral giant cell granuloma: on gingival margin.

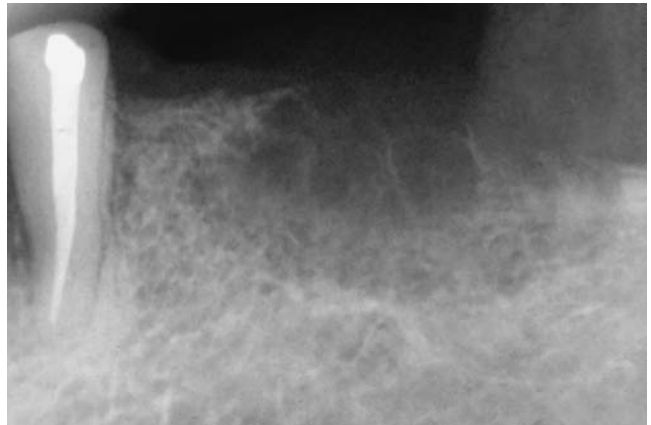


Fig. 40.4. Peripheral giant cell granuloma: "cuffing of bone."



Fig. 40.5. Peripheral ossifying fibroma: typical location.



Fig. 40.6. Peripheral ossifying fibroma: pink and firm.



Fig. 40.7. Peripheral odontogenic fibroma: arising from PDL.

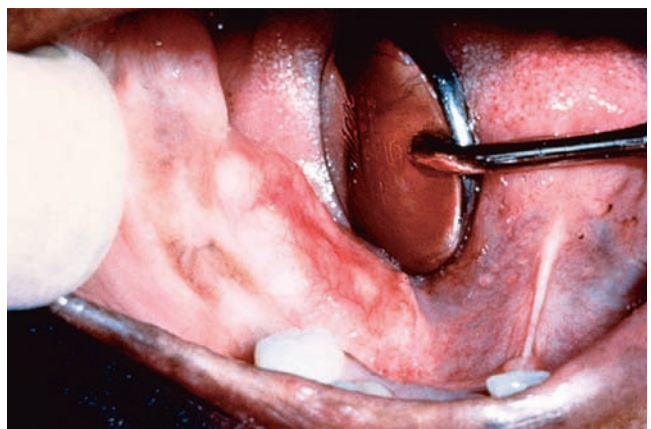


Fig. 40.8. Desmoplastic fibroma: aggressive, firm lesion.

LOCALIZED GINGIVAL LESIONS

Parulis (Gumboil) (Figs. 41.1 and 41.2) The **parulis**, or **gumboil**—the latter term being reserved for children (Fig. 15.8)—is a localized area of inflamed granulation tissue that occurs at the end point of a sinus tract that is draining from a nonvital tooth. It appears as a soft, solitary reddish papule, located apical and facial to a chronically abscessed tooth, usually on or near the labial mucogingival junction. The parulis can be slightly yellow in the center and emit a purulent yellowish exudate on palpation. Acute swelling and pain may accompany the condition if the sinus tract is obstructed.

To locate the nonvital tooth from which the parulis arises, a sterile gutta-percha point may be inserted into the sinus tract. Periapical radiographs are then taken to demonstrate the proximity of the gutta-percha point to the apex of the offending tooth. After the nonvital tooth is diagnosed, the treatment of choice is root canal therapy. Treatment results in healing of the parulis. If the problematic tooth is left untreated, the parulis may persist for years. A persistent lesion may mature into a pink-colored fibroma.

Pericoronitis (Operculitis) (Figs. 41.3 and 41.4) **Pericoronitis** is inflammation of the soft tissue surrounding the crown of a partially erupted or impacted tooth. Pericoronitis may develop at any age but most frequently occurs in children and young adults whose teeth are erupting. Generally, it is associated with an erupting mandibular third molar that is in good alignment but is limited in its eruption by insufficient space. Radiographs reveal a flame-shaped radiolucency in the alveolar bone distal to the tooth, with the cortical outline either absent or distinctly thickened because of infection, deposition of reactive bone, or cyst formation.

Pericoronitis develops from bacterial contamination beneath the **operculum**, resulting in gingival swelling, redness, and halitosis. Pain varies and may be extreme, but the discomfort usually resembles that of gingivitis, a periodontal abscess, or tonsillitis. Regional lymphadenopathy, malaise, and low-grade fever are common. If edema or cellulitis extends to involve the masseter muscle, trismus accompanies the condition. Pericoronitis is frequently complicated by dysphagia (difficulty swallowing) and pain induced by trauma from the opposing tooth during closure.

Pericoronitis is best managed by flushing the purulent material from the gingival sulcus with saline solution and eliminating any occlusal trauma to the operculum from the opposing third molar. Definitive treatment is usually extraction of the involved tooth. Antibiotic coverage is recommended when constitutional symptoms are present and the spread of infection is likely. Recurrences and chronicity are likely if the condition is managed only with antibiotics.

Periodontal Abscess A **periodontal abscess** produces a localized gingival swelling; see Figure 37.7 for discussion.

Epulis Fissuratum (Figs. 41.5 and 41.6) The **epulis fissuratum** is an overgrowth of fibrous connective tissue resulting from chronic irritation usually from the flange of a ill-fitting complete or partial denture. The overextended denture margin initially produces an ulcer that is repeatedly traumatized. Hyperplastic healing results in a pink-red, fleshy exuberance of mature granulation tissue. The hyperplastic lesion, located where the denture flange rests, is found mostly in older women. It is nonpainful, grows slowly on either side of the denture flange, and causes the patient little concern.

The epulis fissuratum in the early stages consists of a single fold of smooth soft tissue. As the swelling grows, a central cleft or several clefts become apparent, the boundaries of which may drape over the denture flange. The mucolabial fold of the anterior maxilla is the most common location, followed by the mandibular alveolar ridge and the mandibular lingual sulcus. Adjustment of the denture, a relin, or construction of a new denture may reduce the trauma and inflammation but will not cause the underlying fibrous tissue to regress. Likewise, surgical excision without alteration of the dentures promotes recurrence. Successful treatment usually requires surgical removal of the redundant tissue, microscopic examination of the excised tissue, and correction or reconstruction of the denture.

Gingival Carcinoma (Figs. 41.7 and 41.8) The gingiva is the site of about 5% to 10% of all cases of oral squamous cell carcinoma. Generally, at the time of diagnosis, the disease is advanced because of its asymptomatic nature, posterior location, and delay in examination.

Gingival carcinoma varies in appearance. It usually appears as a reddish proliferative mass with focal white areas arising from the gingiva that can mimic benign inflammatory and reactive gingival conditions, such as a pyogenic granuloma, erythroplakia, leukoplakia, or a simple ulceration. Carcinoma should be suspected when close examination reveals a pebbly surface, many small blood vessels in the overlying epithelium, and surface ulceration. Etiologic factors include tobacco use, alcoholism, and poor oral hygiene. Elderly men are especially susceptible, and the condition has a slight predilection for the alveolar ridge of the posterior mandible. Completely dentate persons rarely have this disease.

Gingival carcinoma may extend onto the floor of the mouth or mucobuccal fold, or invade the underlying bone. Radiographs may reveal a cupping out of the alveolar crest. Metastasis to regional lymph nodes occurs frequently. Metastatic nodes are firm, rubbery, matted, nonmovable, and nonpainful. Treatment consists of surgery and radiotherapy.



Fig. 41.1. Parulis: reddish papule above nonvital central.



Fig. 41.2. Parulis: adjacent to nonvital first molar.

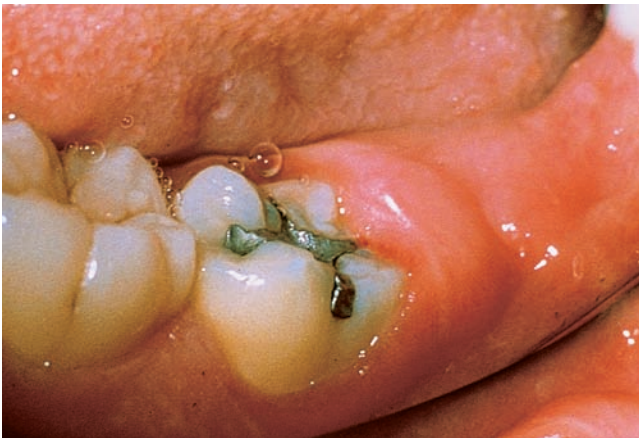


Fig. 41.3. Pericoronitis: distal to partially erupted molar.



Fig. 41.4. Pericoronitis: flame-shaped radiolucency.



Fig. 41.5. Epulis fissuratum: in maxillary labial fornix.



Fig. 41.6. Epulis fissuratum: at partial denture flange.

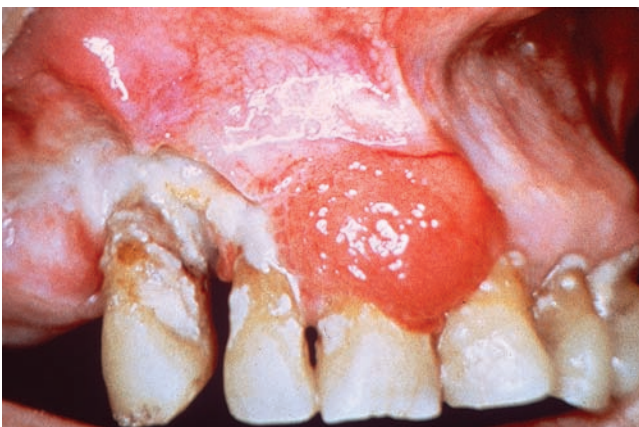


Fig. 41.7. Gingival carcinoma: poor oral hygiene.



Fig. 41.8. Gingival carcinoma: squamous cell type.

GENERALIZED GINGIVAL ENLARGEMENTS

Fibromatosis Gingivae (Figs. 42.1 and 42.2) **Fibromatosis gingivae** is a rare, slowly progressive, fibrous enlargement of the gingiva that may be hereditary or idiopathic. The gingival tissues contain fibroblasts that have low growth activity, dense collagen, and minimal inflammation. The condition begins with tooth eruption and becomes more prominent with age. The enlargement is usually generalized and noninflammatory, affecting the buccal and lingual surfaces of one or both jaws. The free, interproximal, and marginal gingiva are enlarged, uniformly pink, firm, nonhemorrhagic, and often nodular.

There are two types of fibromatosis gingivae: generalized and localized. The generalized type is nodular and diffuse, exhibiting several coalesced areas of globular gingival overgrowths that encroach on and eventually cover the crowns of the teeth. In the less common localized variety, solitary overgrowths are limited to the palatal vault of the maxillary tuberosity or the lingual gingiva of the mandibular arch. These gingival overgrowths appear smooth, firm, and symmetrically round. The localized involvement may be unilateral or bilateral.

Fibromatosis gingivae may interfere with tooth eruption, mastication, and oral hygiene. In severe cases, noneruption of the primary or permanent teeth may be the chief symptom. Regression in dentate patients is not a feature of this disease, even with effective oral hygiene measures. However, in areas of tooth loss, gingival size tends to decrease. Gingivectomy, with either a blade or a carbon dioxide laser, is the usual treatment. Continued growth may require several operations. The condition may be accompanied by acromegalic facial features, hypertrichosis, mental deficits, deafness, and seizures or associated with other syndromes.

Drug-Induced Gingival Overgrowth (Figs. 42.3–42.7) **Gingival overgrowth** is an adverse effect associated with use of specific prescription drugs. It occurs in 25% to 50% of patients taking phenytoin (Dilantin) and cyclosporine (Sandimmune). Phenytoin is an antiseizure medication. Cyclosporine is taken by organ transplant patients to inhibit T-cell proliferation and prevent transplant rejection.

Gingival overgrowth also occurs in 1% to 10% of patients who take calcium-channel blocker drugs (such as nifedipine [Procardia], diltiazem [Cardizem], verapamil [Calan], felodipine [Plendil], and amlodipine [Norvasc]). These drugs relax and dilate artery muscles and are used to lower blood pressure and prevent chest pain (angina) resulting from coronary artery spasm. Sodium valproate (Depakene), an antiseizure medication, and estrogen in birth control pills have also been associated with gingival overgrowth, generally at high doses. Although the mechanism of drug-induced gingival overgrowth remains unknown, many of these drugs affect calcium ion flux in gingival fibroblasts and appear to alter collagen metabolism, collagenase activity, and

the local immune response. Estrogen may work separately by increasing the blood supply and inflammatory mediators to the gingiva. *Note:* Modern low-dose birth control pills rarely cause gingival overgrowth.

Drug-induced gingival overgrowth occurs at any age and in either sex. Although the overgrowth results from a hyperplastic response, an inflammatory component induced by dental bacterial plaque often coexists and tends to exacerbate the condition. The gingival overgrowth is usually generalized and begins at the interdental papillae. It appears most exaggerated on the labial aspects of the anterior teeth. The overgrowths form soft, red, lumpy nodules that bleed easily. Progressive growth results in fibrotic changes: The interdental tissue becomes enlarged, pink, and firm. With time, the condition can completely engulf the crowns of the teeth, which restricts home care, limits mastication, and compromises aesthetics.

The condition can be minimized by instituting excellent patient oral hygiene care upon initiation of drug therapy. Once overgrowth develops, treatment may involve changing drug therapy and the provision of meticulous and frequent plaque control measures. The gingival swelling may completely regress by discontinuing drug use. However, excess fibrotic tissue unresponsive to changes in drug therapy should be surgically removed.

Primary Herpetic Gingivostomatitis (Fig. 42.8) **Herpes simplex virus** is a large DNA virus that infects human epithelium. Transmission is by contact with infected secretions such as saliva. The initial infection is usually subclinical (not readily visible) but can be prominent depending on the amount of initial dose, location, integrity of the epithelium, and host response. **Herpetic gingivostomatitis** is a prominent manifestation of the primary oral infection. Replication of the virus in gingival epithelium causes generalized swelling, redness, and pain of the marginal gingiva. The interdental papillae become bulbous and bleed easily about 4 days after infection, and several oral vesicles and ulcers develop. The virus can spread throughout the oral mucosa and invade peripheral nerve endings. Once inside the infected nerve endings, the virus travels intra-axonally to the trigeminal ganglion, where it enters into a latent state. Systemic antiviral agents such as acyclovir (Zovirax), famciclovir (Famvir), or valacyclovir (Valtrex) are recommended early during the herpetic infection. Antimicrobial mouth rinses with chlorhexidine gluconate 0.12% (Peridex, PerioGard) also help to reduce oral sepsis. Systemic antibiotics are occasionally needed when patients become septic and their temperature is persistently elevated. Healing normally takes about 14 to 21 days. Thereafter, 30% to 40% of latently infected patients develop **recurrent herpes simplex virus infections** that reappear at sites previously infected (see Figs. 72.4–72.6 for more information).



Fig. 42.1. Fibromatosis gingivae: generalized.

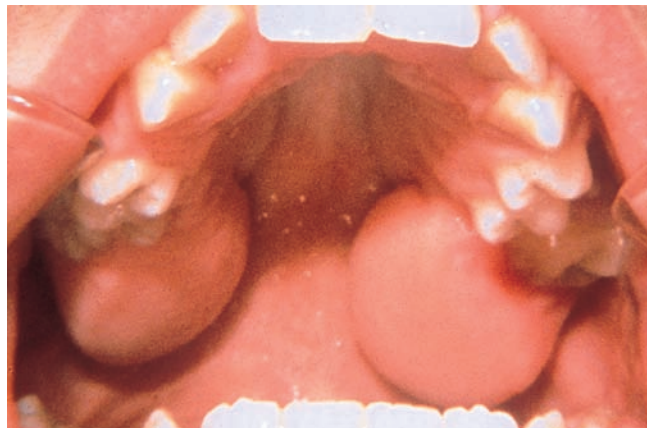


Fig. 42.2. Fibromatosis gingivae: localized variant.



Fig. 42.3. Dilantin-induced gingival overgrowth.



Fig. 42.4. Dilantin-induced gingival overgrowth.

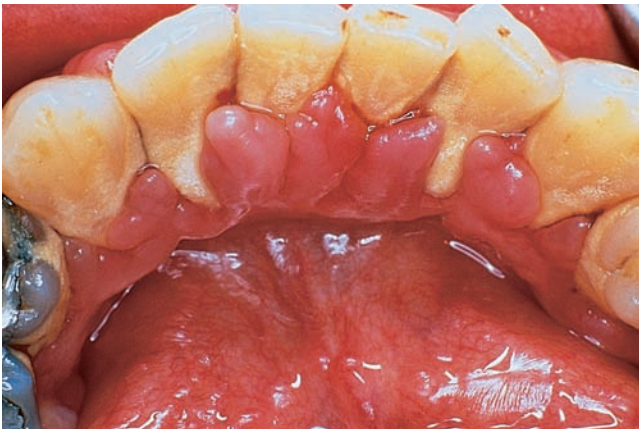


Fig. 42.5. Nifedipine-induced gingival overgrowth.



Fig. 42.6. Nifedipine-induced gingival overgrowth.



Fig. 42.7. Cyclosporine-induced gingival overgrowth.



Fig. 42.8. Primary herpetic gingivostomatitis: painful.

ENDOCRINE-ASSOCIATED GINGIVAL ENLARGEMENTS

Hormonal Gingivitis (Pregnancy Gingivitis) (Figs. 43.1–43.3) **Hormonal gingivitis** is a hyperplastic reaction to microbial plaque that affects women during pregnancy and, to a lesser extent, women during puberty and menopause. Elevated estrogen or progesterone levels resulting from hormonal shifts and use of (previously marketed versions of) birth control pills have been implicated. These hormones enhance tissue vascularity, which permits an exaggerated inflammatory reaction to plaque.

Hormonal gingivitis begins at the marginal and interdental gingiva, usually in the second month of pregnancy. It produces fiery red, swollen, and tender marginal gingiva, and compressible and swollen interdental papillae. The tissue is tender to palpation and bleeds easily on probing. The severity is related to plaque accumulation and poor oral hygiene. This is often the case during pregnancy when toothbrushing may be less frequent because it precipitates nausea.

Hormonal gingivitis is usually transitory and responds to meticulous home care, oral prophylaxis, and a decrease in hormone levels—which occurs after parturition or with a change of hormone medication. Persistence can result in fibrosis and pinker, firmer, and lumpy tissues. In a small percentage of pregnant women, the hyperplastic response may exacerbate in a localized area resulting in a **pyogenic granuloma** (pregnancy tumor) (Fig. 40.2). Gingivectomy is used to reduce fibrotic gingivae and excise tumorous growths.

Diabetic Gingivitis (Figs. 43.4–43.6) **Diabetes mellitus** is a common metabolic disease, affecting approximately 7% of the U.S. population and a high percentage of Hispanic Americans, African Americans, and Pacific Islanders. It is characterized by diminished insulin production (type 1) or diminished insulin use (type 2) that results in poor control of blood glucose levels. Before diagnosis, patients may present with hyperglycemia, glucosuria, polyuria, polydipsia, pruritus, weight gain or loss, loss of strength, visual problems, and loss of peripheral nerve sensation (neuropathy). It can also be a feature of **metabolic syndrome**—a cluster of disorders including insulin resistance, obesity, hypertension, cholesterol abnormalities, and increased risk for clotting. Because diabetes is a progressive disorder affecting large and small blood vessels, complications include many vascular-related problems, including heart disease and stroke, blindness, high blood pressure, kidney failure, risk for infection, dry mouth, burning tongue, and persistent gingivitis.

The severity of **diabetic gingivitis** is dependent on the level of glycemic (blood glucose) control. In the patient with uncontrolled or poorly controlled diabetes, peculiar proliferations of swollen tissue arise from the marginal and attached gingiva. The well-demarcated swellings are soft, red, irregular, and sometimes hemorrhagic. The surface of the hyperplastic tissue is bulbous or lobular. The base of the tissue may be sessile or stalklike. The condition is often accompanied by dry mouth, breath odor changes, and alveolar bone loss because of periodontitis.

The gingivitis is difficult to manage when blood glucose levels remain elevated as the inflammatory response in the periodontal tissues is altered. Successful treatment requires meticulous patient self-care and control of the blood glucose level with diet, hypoglycemic agents, or insulin. *Note:* A well-controlled diabetic patient will have a fasting blood glucose level below 126 mg/dL and a glycosylated hemoglobin (HbA_{1c}) below 8%. Oral surgical procedures should be limited to when the blood glucose level is below 200 mg/dL and the patient's condition is considered stable.

Gingival Edema of Hypothyroidism (Figs. 43.7 and 43.8)

Hypothyroidism is a relatively common disorder that is characterized by low levels of thyroid hormone. The clinical manifestations depend on the age at onset, duration, and severity of thyroid insufficiency. When thyroid hormone (tri-iodothyronine [T₃] or thyroxine [T₄]) deficiency occurs during infancy, **cretinism** results. This disease is characterized by short stature, mental retardation, disproportionate head-to-body size, delayed tooth eruption, mandibular micrognathism, and swollen face, lips, and tongue. Regardless of the age at onset, hypothyroid patients are intolerant to cold and have coarse, yellowish skin that feels cool and dry to the touch, coarse hair, and lethargy. Adult-onset hypothyroidism is also characterized by dull expression, loss of eyebrow hair, slow physical and mental activity, and elevated cholesterol. Soft tissue swelling is the classic feature and is most prominent in the face, particularly around the eyes. It is caused by accumulation of subcutaneous fluid.

Hypothyroidism can be primary or secondary. In primary, the thyroid gland is abnormal or forms incorrectly. Secondary hypothyroidism is due to lack of production of thyroid-stimulating hormone (TSH) by the pituitary gland. Hypothyroidism in children is most commonly caused by primary disease—hypoplasia or agenesis of the thyroid gland. Hypothyroidism in adults is commonly caused by medical therapy that is used to eliminate an overactive thyroid gland (hyperthyroidism), as well as by autoimmune lymphocytic infiltration (**Hashimoto thyroiditis**), in which the thyroid glandular cells are replaced by lymphocytes.

Hypothyroidism can produce an enlarged thyroid gland and oral manifestations. Within the mouth, an enlarged tongue (macroglossia) and enlarged lips (macrocheilia) are common and may cause an altered speech pattern. The gingiva appears uniformly enlarged, pale pink, and compressible. Swelling occurs in all directions on both the facial and lingual sides of the dental arches. When secondary inflammation is present, the tissues become red and boggy and have a tendency to bleed easily. Treatment for the gingival condition depends on the degree of thyroid deficiency. Patients with marginal deficiency require only strict oral hygiene measures, whereas frank cases require supplemental replacement thyroid therapy (thyroxin or levothyroxin) to achieve resolution of the systemic and oral condition.



Fig. 43.1. Hormonal gingivitis: during pregnancy.



Fig. 43.2. Hormonal gingivitis: 3 weeks postpartum.



Fig. 43.3. Hormonal gingivitis: caused by birth control pills.



Fig. 43.4. Diabetic gingivitis: advanced periodontitis.



Fig. 43.5. Diabetic gingivitis: with periodontal abscess.



Fig. 43.6. Diabetic gingivitis: nodular, fiery red.



Fig. 43.7. Hypothyroidism: edema, thick skin.*

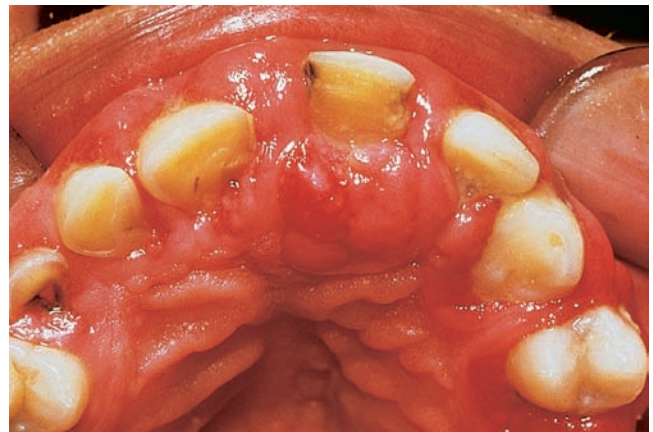


Fig. 43.8. Hypothyroidism: gingival edema.*

SPONTANEOUS GINGIVAL BLEEDING

Leukemic Gingivitis (Figs. 44.1 and 44.2) **Leukemia** is a malignant condition characterized by overproduction of leukocytes and classified by the white blood cell type involved and the clinical course (acute or chronic). Oral manifestations are more frequently encountered in acute leukemia of the monocytic and myelogenous subtypes. Oral features occur early in the course of the disease because of neoplastic proliferation of the white blood cells that deposit in oral tissue. Systemic symptoms develop as the neoplastic cells overwhelm the normal production of the other hematopoietic cells.

Consistent signs of acute leukemia are cervical lymphadenopathy, malaise, anemic pallor, leukopenia-induced ulcerations, recurrent infections, and gingival changes. Multiple adjacent gingival sites are affected and appear red, tender, and spongy. With progression of the disease, the swollen gingiva becomes shiny purple and tends to lift away from the teeth. Stippling of the tissue is lost, and spontaneous bleeding from the gingival sulcus eventually occurs. The edematous tissue is most prominent interdentally and results from leukemic infiltration of malignant cells. In certain patients, the neoplastic cells may invade pulpal and osseous tissue, inducing vague symptoms of pain without corresponding radiographic evidence of pathosis. Purpuric features, such as petechial lesions and ecchymoses on pale mucosal membranes, together with gingival hemorrhage, occur frequently. Systemic control of leukemia often involves intensive radiotherapy, chemotherapy, blood transfusions, and bone marrow transplantation. Difficulty may be encountered in maintaining optimal oral health because of chemotherapy-induced oral ulcerations. Meticulous oral hygiene combined with antimicrobial rinses is recommended to reduce oral inflammation and ulcerations caused by chemotherapy.

Agranulocytosis (Neutropenia). **Agranulocytosis** (or neutropenia) is a disease characterized by a decrease in the number of circulating neutrophils ($<1,500$ cells/mm³)—cells that defend against infection. Most often, the condition is recognized by clinical symptoms, which consist of chronic infections and an almost complete absence of neutrophils in lab blood tests. Cytotoxic drugs and disorders that decrease bone marrow production or destroy neutrophils are causative agents in the majority of cases. Rarely, the condition may be congenital. Uncontrollable infection in the neutropenic patient can result in bacterial pneumonia, sepsis, and death.

Cyclic Neutropenia (Figs. 44.3 and 44.4) A distinct form of agranulocytosis is **cyclic neutropenia**, an inherited disorder characterized by a periodic decrease in circulating neutrophils that occurs about every 3 weeks and lasts for about 5 days. The condition is caused by a inherited (autosomal dominant) mutation in the *ELA2* gene that encodes for neutrophil elastase. Cyclic neutropenia appears in childhood and is sometimes accom-

panied by arthritis, pharyngitis, fever, headache, and lymphadenopathy. A history of repeated infections of the skin, ear, and upper respiratory tract is common. Oral manifestations include periodic inflammatory gingival changes and mucosal ulcerations. The ulcerations are usually large, oval, and persistent. They vary in size and location. Sometimes they are found on the attached gingiva and other times on the tongue and buccal mucosa. At stages that correspond to elevated levels of circulating neutrophils, minimal inflammation is evident. In contrast, when the neutrophil count decreases precipitously, generalized inflammatory hyperplasia and erythema occur. If left untreated, the condition is exacerbated by the presence of local factors, such as plaque and calculus, which result in alveolar bone loss, tooth mobility, and early exfoliation of teeth.

Periodic appearance and spontaneous regression of signs and symptoms is characteristic of cyclic neutropenia. The diagnosis is made by daily measurement of the leukocyte count. Treatment involves use of the blood growth factor known as *granulocyte colony-stimulating factor*, along with antimicrobial agents and a strict oral hygiene program.

Thrombocytopathic and Thrombocytopenic Purpura (Figs. 44.5–44.8)

Platelets help maintain hemostasis by providing the primary hemostatic plug and by activating the intrinsic system of coagulation. A decrease in the number of circulating platelets (**thrombocytopenia**) may be idiopathic or may be the result of decreased platelet production in the bone marrow, increased peripheral destruction, or increased splenic sequestration. Decreased function of circulating platelets (**thrombocytopathia**) is often related to hereditary syndromes or acquired states, such as drug-induced bone marrow suppression, liver disease, or dysproteinemic states like uremia. Mild thrombocytopathia is caused by daily use of aspirin or clopidogrel (Plavix).

The normal platelet count is 150,000 to 400,000 cells/mm³. Vascular-related clinical manifestations of platelet disorders usually do not develop until the count decreases to $<75,000$ cells/mm³. Features include petechiae, ecchymoses, epistaxis, hematuria, hypermenorrhea, and gastrointestinal bleeding resulting in melena. **Spontaneous gingival bleeding** is a frequent, early, and dramatic occurrence. Blood oozes profusely from the gingival sulcus, either spontaneously or after minor trauma, such as toothbrushing. The fluid then turns into purplish black globs of clotted blood that adhere to the oral structures. Clotted blood is sometimes swallowed, which can cause nausea. Mild traumas, particularly at the occlusal line of the buccal mucosa and tongue, are sites of extensive hemorrhage. Red petechial spots on the soft palate is another frequent clinical sign. Measurement of the platelet count and platelet function tests should be done to assess platelet disorders. Platelet transfusions may be necessary if local measures do not control oral bleeding.



Fig. 44.1. Leukemic gingivitis: acute myelogenous leukemia.



Fig. 44.2. Leukemic gingivitis: acute lymphocytic leukemia.



Fig. 44.3. Cyclic neutropenia: gingival erythema.*

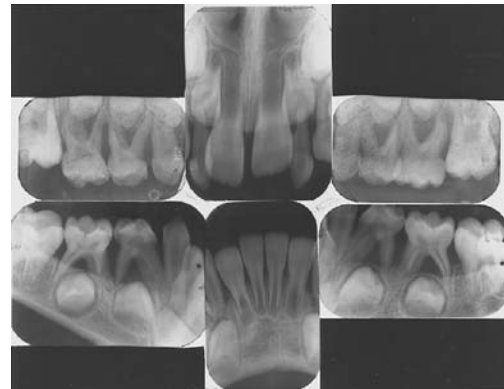


Fig. 44.4. Cyclic neutropenia: floating teeth.*



Fig. 44.5. Thrombocytopathia: and cirrhosis.†



Fig. 44.6. Thrombocytopathia: spontaneous bleeding.†



Fig. 44.7. Thrombocytopenia: 26,000 platelets/mm³.‡

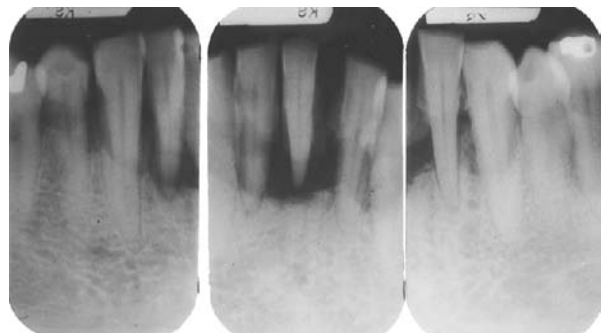


Fig. 44.8. Thrombocytopenia evidence of chronic irritants.³

CASE STUDIES

CASE 13. (Fig. 44.9) This is a 31-year-old patient. He is mentally retarded and takes medication for seizures. His parents are deceased, and he has been institutionalized without proper oral care for several years.

1. Describe the clinical findings.
2. Would these tissues bleed easily? What features provide a clue?
3. What features are suggestive that the condition has been present for some time?
4. What factors are contributing to the condition?
5. What medication is he likely taking?
6. What is abnormal about the teeth and suggestive of his medical condition?
7. What additional gingival abnormalities are present?



CASE 14. (Fig. 44.10) A healthy 7-year-old boy appears at the dental clinic with this soft tissue mass. It has been present for 3 weeks and has progressively increased in size. The patient claims that moderate bleeding occurs every time he brushes his teeth, so he has avoided brushing that area for the last several days. All the adjacent teeth are asymptomatic and test vital. Periapical radiographs of the area reveal no abnormalities.

1. Describe the clinical findings.
2. Is this a normal finding, a variant of normal, or disease?
3. Do you expect this condition to be painful, and if so, why?
4. This lesion shows features of malignancy.
 - A. True
 - B. False
5. The most likely diagnosis for the condition is:
 - A. Leukemic infiltrate
 - B. Parulis
 - C. Peripheral ossifying fibroma
 - D. Pyogenic granuloma
6. List other intraoral sites where this can occur.
7. What are the treatment options for this tooth?
8. What clinical features are suggestive that this is a young patient?



Abnormalities by Location

Dental Objectives:

- Define and use diagnostic terms that describe common lesions and swellings of the lip, tongue, floor of mouth, palate, and face.
- Recognize the causes and clinical features of common lesions and swellings of the lip, tongue, floor of mouth, palate, and face.
- Use the diagnostic process to distinguish similar-appearing lesions and swellings of the lip, tongue, floor of mouth, palate, and face.
- Recommend appropriate treatment options for common lesions and swellings of the lip, tongue, floor of mouth, palate, and face.

Dental Hygiene Objectives:

- Define and use diagnostic terms that describe disorders of the tongue, lip, floor of mouth, palate, and face presented in this section.
- In the clinical setting, document the characteristics of oral conditions or deviations from normal that affect the lip, tongue, floor of mouth, palate, and face, and provide the patient with information about the condition or deviation.
- Identify conditions discussed in this section that (1) require the attention of the dentist and/or (2) affect the delivery of periodontal debridement and oral hygiene measures.

In the figure legends, *[†] denotes the same patient.

CONDITIONS PECULIAR TO THE TONGUE

Scalloped Tongue (Crenated Tongue) (Figs. 45.1 and 45.2) A **scallop**ed tongue is a common finding characterized by a series of indentations on the lateral border of the tongue. The condition is caused by abnormal pressure (e.g., suction) within the mouth of persons who clench or brux their teeth. It is usually bilateral but may be unilateral or isolated to a region where the tongue is held in close contact with the teeth. The abnormal pressure on the tongue imprints a distinctive pattern that appears as depressed ovals that are sometimes circumscribed by a raised, white scalloped border. Causes of scalloped tongue include situations that produce abnormal tongue pressure, such as movement of the tongue against teeth, tongue thrusting, tongue sucking, clenching, bruxing, or an enlarged tongue. The pattern can be accentuated when pressure is applied in areas of **diastemata** (spacing between teeth). A scalloped tongue is seen in normal patients, as well as in association with temporomandibular joint disorders, systemic conditions such as acromegaly and amyloidosis, and genetic disorders such as Down syndrome. The condition is harmless and asymptomatic. Treatment is aimed at habit elimination. A prominent linea alba on the buccal mucosa, a frequent coexisting finding, is caused by negative intraoral pressure associated with tongue sucking in persons who clench or brux their teeth.

Macroglossia (Figs. 45.3 and 45.4) **Macroglossia** involves an abnormally enlarged tongue. To assess tongue size, the tongue should be completely relaxed. The normal height of the dorsum of the tongue should be even with the occlusal plane of the mandibular teeth. The lateral borders of the tongue should contact, but not overlap, the lingual cusps of the mandibular teeth. A tongue that extends beyond these dimensions is enlarged.

Macroglossia is either congenital or acquired. Congenital macroglossia can be caused by idiopathic muscular hypertrophy, muscular hemihypertrophy, benign tumors, vascular malformations, hamartomas, allergic reactions, or cysts. Congenital macroglossia that develops at an early age is a component of Beckwith-Wiedemann syndrome and Down syndrome. Acquired macroglossia may be the result of passive enlargement of the tongue when mandibular teeth are lost. In this case, the enlargement may be localized or diffuse, depending on the size of the edentulous area. Systemic disease, such as acromegaly, amyloidosis, hypothyroidism, or neoplasms, which can occlude lymphatic drainage and produce a swollen tongue, can cause macroglossia. Indicators of an enlarged tongue are focal or diffuse enlargement of the tongue, speech difficulties, displaced teeth, malocclusion, or a scalloped tongue. If the enlarged tongue is hindering function, elimination of the primary cause or surgical correction may be necessary. An enlarged tongue can cause patients to have difficulty accommodating a removable prosthesis.

Hairy Tongue (Lingua Villosa, Coated Tongue) (Figs. 45.5 and 45.6) **Hairy tongue** is an abnormal elongation of the filiform papillae that gives the dorsum of the tongue a hairlike appearance. It is a hypertrophic response associated with increased keratin deposition or delayed shedding of the cornified layer. Patients who do not cleanse their tongues are most commonly affected. Cancer therapy, infection with *Candida albicans*, irradiation, poor oral hygiene, change in oral pH, smoking, and use of antibiotics or oxidizing mouthwashes are associated with this condition.

Hairy tongue may be white, yellow, green, brown, or black, hence the names white-coated tongue and yellow, brown, or black hairy tongue. The color of the lesion is a result of intrinsic factors (chromogenic organisms) combined with extrinsic factors (food, drink [coffee, tea], and tobacco stains). Hairy tongue occurs more frequently in men and primarily in persons older than age 30 years. It increases in frequency with age and consumption of a soft diet. The condition begins in the midline of the tongue near the foramen cecum and spreads laterally and anteriorly. The affected filiform papillae discolor, progressively elongate, and may reach a length of several millimeters. Generally, hairy tongue is only of cosmetic concern, and the tongue remains asymptomatic. Brushing the tongue daily with abrasive pastes along with elimination of predisposing factors leads to resolution. In refractory cases, an underlying condition, such as a dry mouth or diabetes mellitus, should be investigated.

Hairy Leukoplakia (Figs 45.7 and 45.8) **Hairy leukoplakia** is a disorder characterized by raised, white, corrugated lesions on the lateral border of the tongue. It is caused by replication of Epstein-Barr virus within the affected epithelial cells. The lesion is seen almost exclusively in patients infected with human immunodeficiency virus (HIV) or persons with immunosuppression resulting from drugs taken for organ transplantation or systemic disease. The white lesion is primarily located on the lateral borders of the tongue but may extend to cover the dorsal and ventral surfaces, and has been documented on the palate and buccal mucosa. Hairy leukoplakia is so named because hairlike peeling of the parakeratotic surface layer is evident histologically. The fungal organism *Candida albicans* is frequently associated with this lesion.

Early lesions have alternating faint white folds and adjacent normal pink troughs that produce a characteristic vertical white-banded washboard appearance. The bands eventually coalesce to form discrete white plaques or extensive thick white corrugated patches. Large lesions are usually asymptomatic, have poorly demarcated borders, and do not rub off. Bilateral occurrence is common, but unilateral lesions are possible. Antiviral agents that block or limit replication of Epstein-Barr virus are useful in, reducing the size of or, eliminating the lesions. Therapies that return immune health also lead to resolution.



Fig. 45.1. Scalloped tongue: associated with clenching.



Fig. 45.2. Scalloped tongue: caused by tongue sucking.



Fig. 45.3. Macroglossia: congenital hemihypertrophy.



Fig. 45.4. Macroglossia: caused by a hemangioma.



Fig. 45.5. White hairy tongue: from drug therapy.



Fig. 45.6. Brown hairy tongue: after antibiotics.



Fig. 45.7. Hairy leukoplakia: white corrugations in AIDS.



Fig. 45.8. Hairy leukoplakia: seen during dental treatment.

CONDITIONS PECULIAR TO THE TONGUE

Geographic Tongue (Benign Migratory Glossitis, Erythema Migrans) (Figs. 46.1–46.4) **Geographic tongue** is a benign inflammatory condition characterized by irregular patches found mainly on the dorsum of the tongue. The irregular pattern of patches gives the surface of the tongue a map-like appearance, thus the term *geographic*. It occurs in about 1% of the population. Women and young adults are most frequently affected. The cause is unknown, but stress, nutritional deficiencies, and hormonal and hereditary factors may be relevant. The condition classically occurs on the dorsal and lateral surfaces of the anterior two thirds of the tongue, affecting only the filiform and leaving the fungiform papillae intact.

Geographic tongue manifests in three patterns: (1) patchy areas of desquamated filiform papillae; (2) patchy desquamated areas delineated by raised, white, circinate (ring-shaped) borders; and (3) patchy areas of desquamated filiform papillae bordered by an erythematous band of inflammation. Mixtures of these patterns may be present, and the pattern on the surface of the tongue may change in size and location from day to day. Symptoms are uncommon in the first two patterns, but presence of the red inflammatory band is often associated with irritation by spicy foods and reports of burning. The condition may appear suddenly and persist for months or years. Spontaneous remissions and recurrences are typical.

Geographic Stomatitis (Areata Erythema Migrans) (Figs. 46.5 and 46.6) **Stomatitis** is inflammation of the mucous lining of any of the soft tissues of the mouth. Geographic tongue is occasionally seen in association with **geographic stomatitis** and often with fissured tongue. Geographic stomatitis produces red annular patches of the labial and buccal mucosa, soft palate, and occasionally floor of mouth. The patches are mild erosions of the mucosa. When asymptomatic, geographic tongue or stomatitis requires no treatment. Symptomatic lesions generally respond to topical anesthetics or topical steroids, in combination with stress reduction.

Anemia (Fig. 46.7) **Anemia** is a condition characterized by impaired oxygen delivery to body tissue as a result of a reduction in the number of erythrocytes, hemoglobin (the protein within the red blood cells that carries oxygen), or total blood volume. Underlying causes of anemia include decreased production of erythrocytes caused by a nutritional deficiency state or bone marrow suppression, increased destruction of erythrocytes, or increased blood loss as a result of hemorrhage. Iron deficiency is the most common type of anemia, frequently affecting middle-aged women and young teenagers. It produces a microcytic (small red blood cells) anemia.

Deficiencies in vitamin B₁₂ and folic acid cause macrocytic anemia (large red blood cells).

Anemia is a sign of an underlying disease. Thus, the cause of anemia must always be sought. Anemia produces changes in the oral mucosal membranes, but these alterations are not helpful in distinguishing the type of anemia. Analysis of erythrocyte indices is required for an accurate diagnosis. Intraoral manifestations of anemia are most prominent on the tongue. The dorsum of the tongue initially appears pale, with flattening of the filiform papillae. Continued atrophy of the papillae results in a surface that is devoid of papillae and appears smooth, dry, and glazed. This condition is commonly called bald tongue. In the final stage, a beefy or fiery red tongue is seen, sometimes with concurrent oral aphthae.

Anemia may produce a sore, painful (**glossodynia**), or burning tongue (**glossopyrosis**). The lips may be thinned and taut, and the width of the mouth may develop a narrowed appearance. Other clinical signs associated with anemia include angular cheilitis, aphthae, dysphagia, mucosal erythema and erosions, pallor, shortness of breath, fatigue, dizziness, a bounding pulse, tingling of the extremities, or difficulty walking. Therapy should be directed toward correcting the underlying cause. After therapy the appearance of the oral tissue improves.

Fissured Tongue (Fig. 46.8) **Fissured tongue** is a relatively common condition that presents as numerous linear grooves or fissures on the dorsum of the tongue. In children, it is often associated with inherited disorders and is a component of **Melkersson-Rosenthal syndrome** (fissured tongue, swollen lips, and facial paralysis; Figs. 50.4–50.6). In adults, it is commonly associated with **xerostomia**. Many therapeutic drugs—primarily antidepressant agents, antihistamines, antihypertensive and cardiac agents, decongestants, ganglionic blocking agents, and tranquilizers—produce xerostomia and thus fissured tongue.

Fissured tongue can have a varied appearance. Many cases have a prominent midline groove and several branching lateral grooves. Others have multiple irregular, wavy grooves. The fissures are often 2 to 5 mm deep and have varying widths that narrow toward the periphery. Interspersed between the fissures are islands of papillae that may appear dry, atrophic, or geographic. There is a strong association with geographic tongue. Most patients are asymptomatic, although some may report mild discomfort or burning. The condition is benign and does not require treatment. However, patients should be encouraged to brush the affected regions to minimize food and bacterial accumulations.

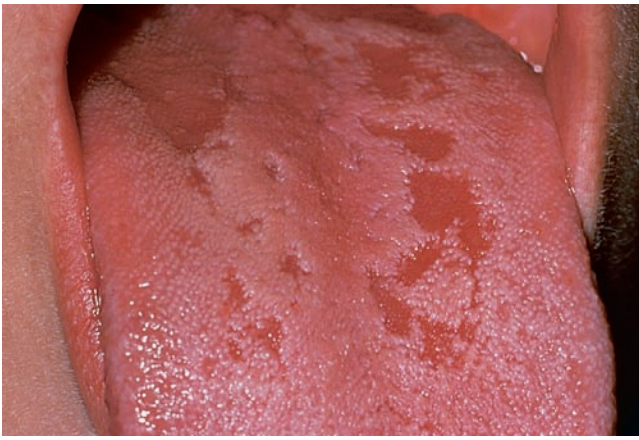


Fig. 46.1. Geographic tongue: asymptomatic denuded areas.



Fig. 46.2. Geographic tongue: asymptomatic white regions.



Fig. 46.3. Geographic tongue: red and painful.



Fig. 46.4. Geographic tongue: red and painful.

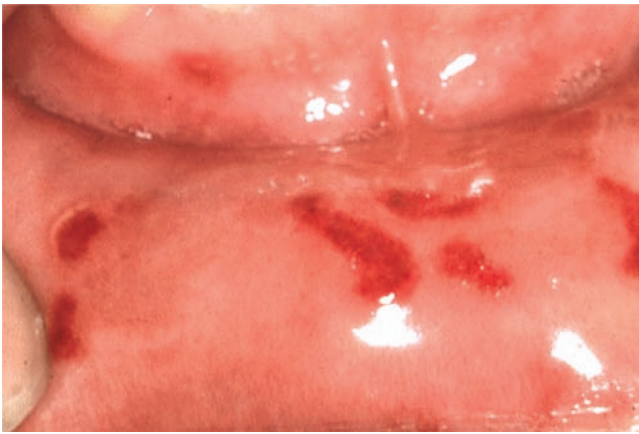


Fig. 46.5. Geographic stomatitis: symptomatic labial mucosa.



Fig. 46.6. Geographic stomatitis: annular pattern on palate.



Fig. 46.7. Iron deficiency anemia: bald, burning tongue.

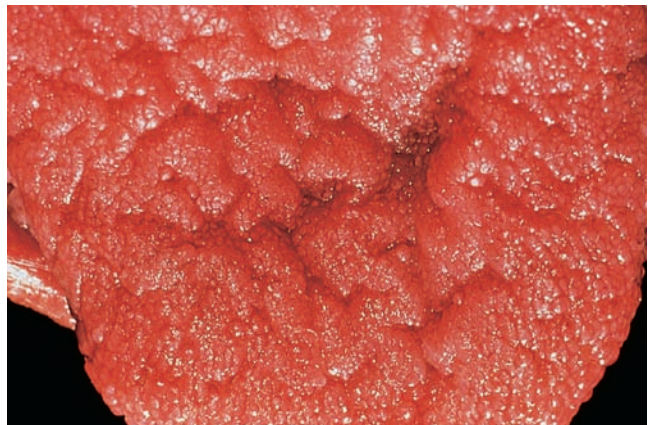


Fig. 46.8. Fissured tongue: dry, fissured, and atrophic.

CONDITIONS PECULIAR TO THE TONGUE

Cyst of Blandin-Nuhn (Lingual Mucus-Retention Phenomenon) (Fig. 47.1) The glands of **Blandin-Nuhn** are accessory salivary glands on the ventral surface of the tongue composed of mixed serous and mucous elements. The cyst develops when trauma to the ventral tongue induces leaking of saliva into the surrounding tissues. It appears as a small painless pink-red swelling with raised and well-demarcated borders. The lesion is soft and fluctuant. When superficial, the cyst has balloonlike features and a pedunculated base. Deeper lesions have sessile bases. Although usually induced by trauma, the cyst of Blandin-Nuhn may be congenital. The congenital variant may represent a true epithelial-lined salivary duct cyst. These cysts rarely exceed 1 cm in diameter. Treatment is excisional biopsy.

Median Rhomboid Glossitis (Figs. 47.2–47.4) Median rhomboid glossitis was once thought to be a developmental defect of incomplete descent of the tuberculum impar. This theory is no longer valid. It is now accepted that **median rhomboid glossitis** is an asymptomatic rhomboid- or ovoid-shaped red area on the tongue that results from a chronic *C. albicans* infection. The most common location is the midline of the dorsum of the tongue, just anterior to the circumvallate papillae. The size and shape varies, but frequently it appears as a well-defined, 1- to 2.5-cm red lesion with irregular but rounded borders. It affects middle-aged persons and rarely affects children. There is no racial predilection. The prevalence is often higher in patients who have diabetes, are immunosuppressed, or have completed a course of broad-spectrum antibiotics.

Median rhomboid glossitis presents early in its course as a smooth, denuded, beefy red patch devoid of filiform papillae. With time, the lesion becomes granular and lobular. An erythematous palatal candidal lesion is sometimes observed directly over the lesion of the tongue. In this case, the condition is termed **chronic multifocal candidiasis** (Figs. 47.3 and 47.4).

Median rhomboid glossitis is easily recognized by its clinical appearance, characteristic location, and asymptomatic nature. Early recognition and treatment with antifungal agents usually leads to resolution. End-stage median rhomboid glossitis is usually asymptomatic and refractory to antifungal treatment as the lesion becomes fibrotic and hypovascular.

Granular Cell Tumor (Figs. 47.5 and 47.6) The **granular cell tumor** is a rare, benign, soft tissue tumor composed of polygonal cells that have an extremely granular cytoplasm. This tumor may occur in cutaneous, mucosal, and visceral sites, but about 50% of cases occur in the dorsal-lateral surface of the tongue. Its histogenesis is controversial, but most experts believe that it is a benign proliferation of neural or endocrine cells.

The granular cell tumor can occur at any age and in any race, but it has a slight predilection for women. Usually the lesion consists of an asymptomatic, solitary, dome-shaped submucosal nodule covered clinically by normal, yellow, or white tissue. The surface is generally smooth but may be ulcerated if traumatized. The tumor is often sessile, well circumscribed, and firm to compression. Growth is very slow and painless; some tumors grow to several centimeters in size. Larger lesions may demonstrate a slightly depressed central area. In rare cases, these lesions are found on the ventral surface of the tongue or buccal mucosa. Approximately 10% of affected patients experience multiple lesions.

This tumor is characterized by pseudoepitheliomatous hyperplasia overlying the granular neoplastic cells. In some cases, this hyperplasia may mistakenly resemble epidermoid carcinoma. Conservative local excision is the preferred treatment. These lesions do not tend to recur.

Lingual Thyroid (Fig. 47.7) **Lingual thyroid** is an uncommon nodule of thyroid tissue found just posterior to the foramen cecum on the posterior third of the tongue. It occurs when embryonic tissue from the thyroid gland fails to migrate to the anterolateral surface of the trachea. Persistent thyroid tissue occurs much more frequently in women than in men (the ratio is 4:1) and may appear at any age. If the remnant tissue becomes cystic, the condition becomes a **thyroglossal duct cyst**.

Lingual thyroid is a raised asymptomatic mass that is usually about 2 cm in diameter. Increased surface vascularity is a prominent feature. Symptoms of dysphagia, dysphonia, or hypothyroidism most often develop during puberty, pregnancy, or menopause. Clinicians can differentiate this lesion from similar lesions by confirming its distinctive location posterior to the circumvallate papillae and by using uptake studies of radioactive iodine. Biopsy should be deferred until it is ascertained that the rest of the thyroid gland is present and functioning. In more than 50% of patients with ectopic thyroid, the lingual thyroid is the only active thyroid tissue present.

Body Piercing (Oral Jewelry) (Fig. 47.8) **Body piercing** is the placement of a foreign object through tissue of the body. Common extraoral locations include the ears, eyebrows, ala of the nose, and belly button. Intraorally, the mucobuccal fold of the lower lip and the tongue are common sites. A popular object placed in the pierced tongue is the metallic barbell. Body piercing may be performed without anesthesia and minor regard for infection control. Common adverse events associated with piercing include infection, swelling, bleeding, allergic reactions, tooth fractures, and mucogingival defects. Oral jewelry needs to be removed before taking radiographs, and patients should be advised of the potential oral health problems associated with these objects.



Fig. 47.1. Cyst of Blandin-Nuhn: variant of a mucocele.



Fig. 47.2. Median rhomboid glossitis: typical presentation.

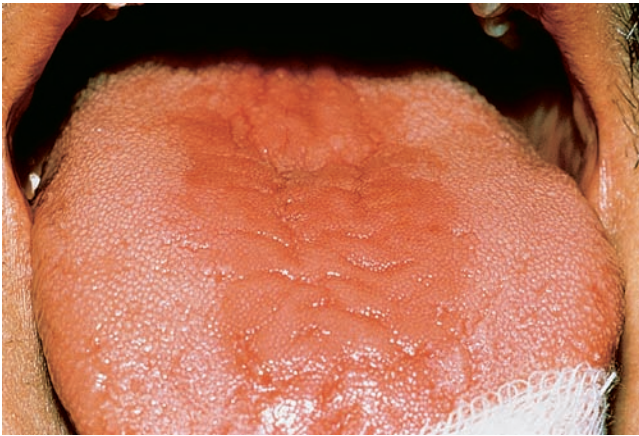


Fig. 47.3. Median rhomboid glossitis: smooth denuded patch.*



Fig. 47.4. Contact palatal lesion: red irregular border.*



Fig. 47.5. Granular cell tumor: pink tongue nodule.†



Fig. 47.6. Granular cell tumor: raised appearance evident.†

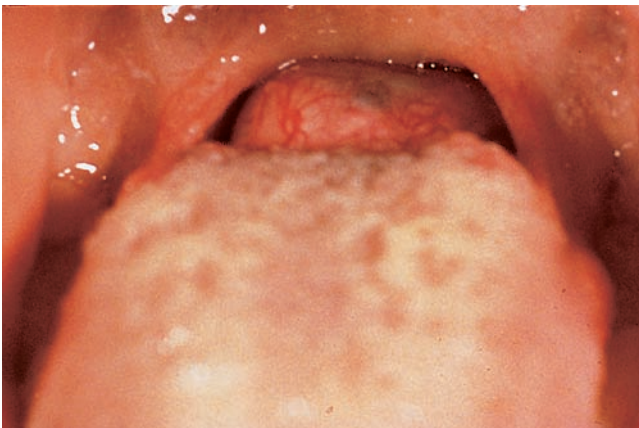


Fig. 47.7. Lingual thyroid: vascular midline mass tongue.

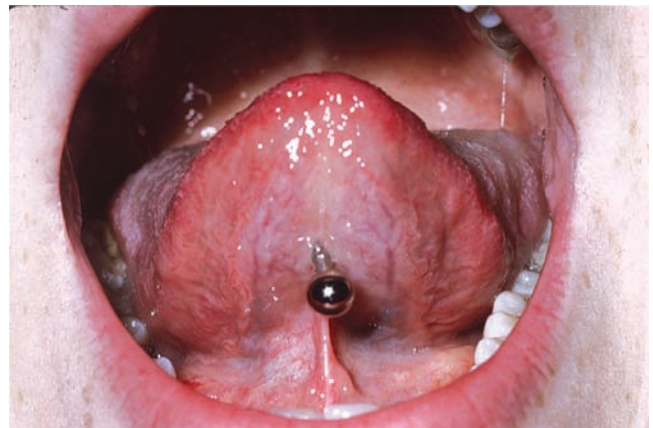


Fig. 47.8. Body piercing: tongue barbell.

CONDITIONS PECULIAR TO THE LIP

Actinic Cheilosis (Actinic Cheilitis) (Figs. 48.1 and 48.2) **Actinic cheilosis** is a premalignant lesion of the vermilion portion of the lower lip caused by excessive exposure to sunlight. Older, fair-skinned men with outdoor occupations are typically affected. In early stages, the lower lip is red and atrophic with subtle blotchy intervening pale areas and loss of the the vermilion border of the lip. With increased sun exposure, irregular scaly areas develop that may thicken and contain focal white patches that can be peeled off. The lip slowly becomes firm, slightly swollen, fissured, and everted. Ulceration with a thin yellow surface crust is typical of the chronic condition. The ulcers may be caused by trauma, loss of elasticity, or may be an early sign of dysplastic or carcinomatous transformation. Histologic features include epithelial atrophy, subepithelial basophilic degeneration of collagen, and increased elastin fibers. Biopsy is recommended to rule out similar sun-related diseases, such as epithelial dysplasia, carcinoma in situ, basal cell carcinoma, squamous cell carcinoma, malignant melanoma, keratoacanthoma, cheilitis glandularis, and herpes labialis.

Up to 10% of actinic cheilosis cases develop into cancer. Clinicians should warn the patient of the likelihood of disease progression without the use of sunscreen protective agents. Dysplastic changes should be treated surgically or with topical applications of 5-fluorouracil.

Candidal Cheilitis (Figs. 48.3 and 48.4) **Candidal cheilitis** is an inflammatory condition of the lips caused by *C. albicans* (a fungal infection) and a lip-licking habit. In its typical presentation, candidal organisms obtain access to and invade the surface layers of the lip after mucosal breakdown, which is caused by repeated wetting and drying of the labial tissues. Desquamation and fissuring of surface epithelium results, and a fine whitish scale consisting of dried salivary mucus may be seen. In children, the affected perilabial skin appears red, atrophic, and fissured. Chapped, dry, itchy, burning lips and the inability to eat hot spicy foods are frequent symptoms. The chronic phase of this infection is characterized by painful vertical fissures that ulcerate and are slow to heal. Nystatin ointment is helpful in resolving the condition, but ultimate resolution requires elimination of the lip-licking habit. In persistent cases, an underlying condition (e.g., chronic use of corticosteroids) or systemic problem (e.g., diabetes mellitus or HIV infection) should be ruled out. A hypersensitivity reaction to ingredients contained in lip balms or lipsticks may clinically mimic this condition.

Angular Cheilitis (Perlèche) (Figs. 48.5 and 48.6) **Angular cheilitis** is a painful condition consisting of radiating erythematous fissures at the corners of the mouth. The condition is most commonly seen after the age of 50

years and is usually encountered in women and denture wearers. Infection by *C. albicans*, *Staphylococcus aureus*, or both is causative. These pathogens are carried to the corners of the mouth from repeated pooling of saliva and frequent licking of the corners of the mouth (perlèche: *to lick* in French). Patients often perform this unconscious habit in an effort to provide relief to the area.

Angular cheilitis initially produces soft, red, and ulcerated mucocutaneous tissue at the corners of the mouth. With time, the erythematous fissures become deep and extend several centimeters from the commissure onto the perilabial skin, or ulcerate and involve the labial and buccal mucosa. The ulcers frequently develop crusts that split and reulcerate during normal oral function. Small yellow-brown granulomatous nodules may eventually appear. Bleeding is infrequent.

Angular cheilitis is chronic and usually bilateral and is often associated with denture stomatitis or glossitis. Predisposing conditions include anemia, poor oral hygiene, frequent use of broad-spectrum antibiotics, decreased vertical dimension, high sucrose intake, dry mouth, accentuated perioral folds, and vitamin B deficiency. Treatment should include preventive measures (such as elimination of traumatic factors, meticulous oral hygiene, re-establishment of the correct vertical dimension and salivary flow) combined with topical antifungal and antibiotic therapy. Vitamin supplementation may also prove beneficial. Elimination of any lip-licking habit is also part of the management protocol.

Exfoliative Cheilitis (Figs. 48.7 and 48.8) **Exfoliative cheilitis** is a persistent condition affecting the lips that is characterized by fissuring, desquamation, and the formation of hemorrhagic crusts. *C. albicans*, oral sepsis, stress, habitual lip-licking and biting, and contact allergens are etiologic agents. An association with psychological and thyroid disorders has been reported. This condition usually begins as a single fissure near the midline of the lower lip and spreads to produce multiple fissures. The fissures may ultimately develop a yellow-white scale or ulcerate and form hemorrhagic crusts over the entire lip. The condition is often bothersome and unsightly, with the lower lip demonstrating more prominent signs. When the condition is symptomatic, burning is the usual chief symptom. Exfoliative cheilitis has a predisposition for teenage girls and young women, and stress has been reported to cause acute exacerbations. Because the cause of the condition appears to be multifactorial, exfoliative cheilitis is difficult to manage and may persist for many years. Treatment is best rendered through the elimination of predisposing systemic or psychological factors together with topical application of antifungal ointments.



Fig. 48.1. Actinic cheilosis: vermilion border lost and scaly.



Fig. 48.2. Actinic cheilosis: everted, thickened with crusts.



Fig. 48.3. Candidal cheilitis: whitish scale is dried mucin.



Fig. 48.4. Candidal cheilitis: in a patient with uncontrolled diabetes.



Fig. 48.5. Angular cheilitis: flaccid perioral folds.*

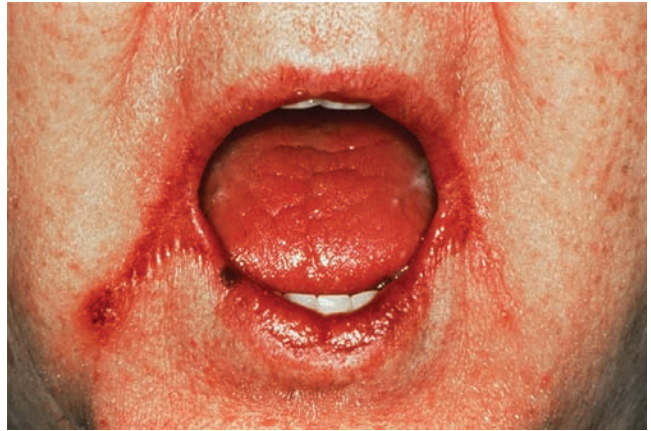


Fig. 48.6. Angular cheilitis: in older adult.*



Fig. 48.7. Exfoliative cheilitis: early lesion; single fissure.



Fig. 48.8. Exfoliative cheilitis: hemorrhagic crusts.

NODULES OF THE LIP

Mucocele (Mucus Extravasation Phenomena) (Figs. 49.1 and 49.2) A **mucocele** is a swelling of the lip or mucosa that is due to mucus from accessory salivary glands of the mouth leaking into the soft tissue when a salivary gland duct is severed. Most mucoceles occur in mandibular labial mucosa. They lack an epithelial lining (so they are not a true cyst) and are surrounded by granulation tissue. The **ranula** is a variant mucocele of the floor of the mouth caused by trauma to a sublingual gland duct (Bartholin's duct) or, rarely, Wharton's duct of the submandibular gland. Mucoceles are distinguished from the rare **salivary duct cyst (mucous retention cyst)**, which is a true cyst with an epithelial lining (Fig. 47.1).

The mucocele constitutes the most common nodular swelling of the lower lip. These swellings are asymptomatic, soft, fluctuant, bluish gray, and usually <1 cm in diameter. Enlargement coincident with meals may be an occasional finding. The most common location is the lower lip midway between the midline and commissure, but other locations include the buccal mucosa, palate, floor of the mouth, and ventral tongue. Children and young adults are most frequently affected. Trauma is the etiologic agent.

Superficial mucoceles often regress spontaneously, whereas deep-seated mucoceles tend to persist and exacerbate with repeated trauma. Persistent mucoceles are treated by surgical excision. Recurrence is possible if the injured accessory salivary glands are not removed or if other ducts are severed during the procedure.

Accessory Salivary Gland Tumor (Figs. 49.3 and 49.4) Nodular swellings of the upper lip are infrequent and are usually caused by **benign minor salivary gland tumors**. About 90% of salivary tumors affect the major glands, and only 10% affect the minor glands. The most common types of benign salivary gland tumors are the **canalicular adenoma** and **pleomorphic adenoma**. These tumors are characterized by encapsulation, slow growth, and long duration (several months). Persons older than age 30 years are more commonly affected. Clinically, the pleomorphic adenoma is a pink to purple dome-shaped or multinodular lesion that protrudes from the inner aspect of the lip or vestibule. It is usually semisolid, freely movable, painless, and especially firm on palpation. The border is well circumscribed. Although it has unlimited potential for growth, the tumor generally remains <2 cm in diameter. Fluctuance and surface ulceration are not usual clinical features.

Malignant salivary gland tumors, such as **mucoepithelioid carcinoma** and **adenocarcinoma**, are rare in the upper lip and may be distinguished from benign neoplasia by their rapid and aggressive growth, short duration, and tendency to ulcerate and cause neurologic symptoms. Treatment of salivary gland neoplasia consists of surgical excision. If the excision is incomplete, recurrences are possible.

Nasolabial Cyst (Nasoalveolar Cyst) (Figs. 49.5 and 49.6) The **nasolabial cyst** is a developmental cyst of soft tissue located in the cuspid–lateral incisor region of the upper lip. The cause is uncertain, and two theories have been suggested. The most accepted theory is that epithelial remnants become entrapped during the embryologic fusion of the lateral nasal, globular, and maxillary processes. A more recent theory suggests that the tissue originates from the nasolacrimal duct. Proliferation and cystic degeneration of the entrapped tissue usually do not become clinically evident until after age 30 years, even though the tissue has been entrapped since birth. The condition has a female predilection.

The nasolabial cyst is a palpable swelling of the upper lip often extending to the M-B fold that can cause elevation of the ala of the nose, dilatation of the nostril, and alteration of the nasolabial fold. The intraoral cyst may be tense or fluctuant, depending on size, and can affect the fit of a maxillary denture. Aspiration yields a yellowish or straw-colored fluid. The cyst is most often unilateral and is generally not in contact with the adjacent bone. Thus, the maxillary teeth remain vital. Infrequently, if the nasolabial cyst applies pressure to the adjacent bone, local resorption of osseous structures can result. Treatment is simple excision.

Implantation Cyst (Epithelial Inclusion Cyst) (Fig. 49.7) An **implantation cyst** is an unusual cyst arising from a foreign-body reaction to surface epithelium that is implanted within epidermal structures after traumatic laceration. The cyst can occur intraorally or extraorally, at any age, and in any race or sex. Within the mouth, the lesion appears as a firm, dome-shaped, freely movable nodule located at the site of impetus, which is often the lip. Implantation cysts are usually small, solitary, and asymptomatic. Growth appears to remain constant, and the overlying mucosa appears smooth and pink. There is no swelling at mealtimes, nor is there spontaneous drainage of mucin as seen in mucocele. A history of trauma should also lead the clinician to suspect this lesion. Surgical excision and histopathologic examination are recommended.

Mesenchymal Nodules and Tumors (Fig. 49.8) A variety of mesenchymal tumors, such as fibroma, lipofibroma, and neuroma, can cause nodular swellings of the lip. The example shown here is a **neurofibroma**. Neurofibromas may be solitary or found in conjunction with von Recklinghausen disease (Figs. 70.7 and 70.8). When solitary, the neurofibroma is usually an asymptomatic, sessile, smooth-surfaced nodule of the buccal mucosa, gingiva, palate, or lips. Histologic examination of the tumor shows connective tissue and nerve fibrils. The discovery of a solitary neurofibroma requires close examination for multiple neurofibromatosis because the latter condition is associated with a marked tendency toward malignant transformation.



Fig. 49.1. Mucocoele: small bluish superficial swelling.



Fig. 49.2. Mucocoele: bluish larger and deeper lesion.



Fig. 49.3. Pleomorphic adenoma: a firm bluish nodule.



Fig. 49.4. Canalicular adenoma: purplish labial nodule.



Fig. 49.5. Nasolabial cyst: a fluctuant nodule on palpation.



Fig. 49.6. Nasolabial cyst: injected with contrast medium.



Fig. 49.7. Implantation cyst: from trauma.



Fig. 49.8. Neurofibroma: sessile base, normal color.

SWELLINGS OF THE LIP

Angioedema (Figs. 50.1 and 50.2) **Angioedema** is a swelling of fluid beneath the skin that is usually the result of an allergic (hypersensitivity) reaction. It occurs in hereditary and acquired forms and may be generalized or localized. Most cases are acquired and result from exposure to an allergen (such as food, cosmetics, latex, or stress) that attracts mast cells that degranulate and release histamine. Infections and autoimmune disease can induce angioedema by triggering capillary permeability via the formation of antigen-antibody complexes or by elevating numbers of eosinophils. Angiotensin-converting enzyme drugs used in the treatment of hypertension can also cause angioedema by increasing bradykinin levels. Histamine and bradykinin mediate capillary permeability and the leakage of plasma into the soft tissues.

The swelling develops within minutes or gradually over a few hours. When swelling affects the lips, it is usually uniform and diffuse but may be asymmetrical. The vermilion appears stretched, everted, pliable, and less distinct; the surface epithelium remains normal in color or is slightly red. Swellings of the tongue, floor of the mouth, eyelids, face, and extremities may accompany the condition. Acquired angioedema is usually recurrent and self-limiting and poses little threat to the patient. Symptoms are limited to burning or itching. Management involves prescription of antihistamines, identification and withdrawal of the allergen, and stress reduction.

The two types of rare hereditary form of angioedema (type I and type II) are autosomal dominant. They cause angioedema by activating the complement pathway. Pharyngeal and laryngeal involvement are usual and may be life-threatening. Hereditary angioedema responds poorly to epinephrine, corticosteroids, and antihistamines. Management involves avoidance of violent physical activity and trauma. Androgenic drugs, such as Danocrine (Danazol), help to prevent attacks.

Cheilitis Glandularis (Fig. 50.3) **Cheilitis glandularis** is a chronic inflammatory disorder of the labial salivary glands that most frequently affects older men. The lower lip is particularly susceptible and is characterized by diffuse enlargement and eversion of the lip. Although the cause remains poorly understood, the condition is associated with chronic exposure to the sun and wind and, less frequently, with smoking, poor oral hygiene, bacterial infection, and hereditary factors.

Clinical manifestations of cheilitis glandularis include a symmetrically enlarged, everted, and firm lower lip. With time, the inflamed labial salivary glands enlarge, and the ductal openings become dilated and appear as multiple small red spots. From these openings a viscous, yellowish, mucopurulent exudate is secreted that makes the lip sticky. Progression of the condition causes the lip to appear atrophic, dry, fissured, and scaly and to become painful. Distinction of the vermilion border is eventually

lost, and secondary infection of a deep labial fissure often results in fistulation and scarring. Emollients and sunscreens afford protection. Severe cases require vermilionectomy (lip shave), which produces an excellent aesthetic result. These patients are at an increased risk for malignant transformation to squamous cell carcinoma.

Orofacial Granulomatosis (Cheilitis Granulomatosa) (Figs. 50.4–50.6)

Orofacial granulomatosis is a condition that results in nonpainful swelling of the orofacial tissues with a histologic finding of granulomatous inflammation in the tissues. The condition has two clinical variants: **cheilitis granulomatosa**, which involves only the lips, and **Melkersson-Rosenthal syndrome**, which has the features of unilateral facial paralysis, fissured pebbly tongue, and persistent labiofacial swelling. The condition appears to represent an abnormal immune reaction to infection or a foreign material/allergen. There is no sex predilection. The labial swelling develops slowly at a young age. Both lips may be firm and swollen, but symmetric enlargement of the lower lip is more common. The diffuse enlargement is asymptomatic and does not affect the color of the lip, but discrete nodules can often be palpated. The tongue, buccal mucosa, gingiva, palatal mucosa, and face can be affected. Patients respond to elimination of the allergen or odontogenic infection, as well as intralesional steroid injections. Spontaneous regression is possible.

Trauma (Fig. 50.7) **Trauma** to the lips often results in edema that is fluctuant, irregular, and exquisitely painful. Trauma may originate from an external source or may be self-induced. The trauma may damage the soft tissue of the lip, resulting in laceration or hemorrhage. Tooth fracture may accompany the condition. Traumatic enlargement of the lip is often a problem of children and mentally disabled patients who inadvertently chew their lip while under local anesthesia. For lip injuries it is best to limit traumatic influences, apply ice compresses, and treat any lacerations or hemorrhage promptly.

Cellulitis (Fig. 50.8) **Cellulitis** means inflammation of cellular tissue. This degenerative process is caused by a bacterial infection in which the localization of pus has yet to occur. When of dental origin, cellulitis typically produces grossly swollen facial tissue that is warm and painful to touch and is extremely hard to palpation. A firm, diffusely swollen lip may be the first sign of cellulitis of odontogenic origin in the anterior region. A nonvital tooth is usually the root of the problem. Failure of host defense mechanisms to control the infection can result in an abscess or spread of infection. Treatment involves removal of necrotic pulpal tissue, drainage of the infection, culture, antibiotic sensitivity testing, and antibiotic therapy. Injection of local anesthetic into the inflammatory region should be avoided to minimize spread of the infection.



Fig. 50.1. Angioedema: both lips swollen and everted.



Fig. 50.2. Angioedema: unilateral upper and lower lip swelling.



Fig. 50.3. Cheilitis glandularis: everted lip; red spots.



Fig. 50.4. Orofacial granulomatosis: facial paralysis.*



Fig. 50.5. Orofacial granulomatosis: lip involvement.*



Fig. 50.6. Orofacial granulomatosis: fissured tongue.*



Fig. 50.7. Trauma: swollen, ulcerated upper lip.



Fig. 50.8. Cellulitis: lip swelling; abscessed incisor.

SWELLINGS OF THE FLOOR OF THE MOUTH

Dermoid Cyst (Figs. 51.1 and 51.2) A **dermoid cyst** is a developmental cyst, usually present at birth, that develops from all three germ layers (teratoma) and is lined by epidermal cells. It may occur anywhere on the skin but has a propensity for the floor of the mouth. Few appear very early in life, and most occur before 35 years of age. There is no sex predilection. Those that arise above the mylohyoid muscle appear as a painless midline, dome-shaped floor of the mouth swelling. The overlying mucosa is a natural pink, the tongue is slightly elevated, and palpation yields a doughlike consistency. Patients may report difficulties in eating and speaking. Growth is slow, but diameters in excess of 5 cm may be seen. Dermoid cysts may appear below the floor of the mouth if the original site of development is inferior to the mylohyoid muscle. In this instance, a submental swelling is noted. The lesion is histologically distinguished from an epidermoid cyst by the presence of adnexal structures, such as sebaceous glands, sweat glands, and hair follicles in the fibrous wall. The lumen contains semisolid keratin and sebum, which accounts for the doughy consistency and makes aspiration difficult. Surgical removal is the preferred treatment.

Ranula (Mucocoele of the Sublingual Gland) (Figs. 51.3 and 51.4) **Ranula** refers to large mucocoeles of the floor of the mouth. Like other mucocoeles, a ranula is caused by pooling of saliva within submucosal tissue and arises from a sialolith or after trauma to a salivary gland duct. Most involve the major excretory duct of the sublingual gland (ducts of Bartholin) or submandibular gland (Wharton's duct). Less commonly, they arise from severed ducts of accessory salivary glands in the floor of mouth. No sex predilection is apparent, and persons younger than 40 years of age are most commonly affected.

There are two types of ranulae: the more common **superficial ranula** that appears as a soft compressible swelling rising up from the floor of mouth and the dissecting or **plunging ranula** that penetrates below the mylohyoid muscle to produce a submental swelling. The superficial ranula is characteristically dome shaped, translucent or bluish, fluctuant, and lateral to the midline. As the asymptomatic lesion enlarges, the mucosa becomes stretched, thinned, and tense. Unlike a dermoid cyst, digital pressure does not cause the lesion to pit. However, it can rupture, causing mucus to escape. The entire floor of the mouth may be filled by the swelling, which elevates the tongue and hinders movement. This impairs mastication, deglutition, and speech.

A ranula should be differentiated from other floor-of-the-mouth swellings by use of sialography, magnetic resonance imaging, and biopsy. Initial treatment is excision or marsupialization (Partsch operation), which consists of excising the overlying mucosa and suturing the remaining cystic lining to the floor of the mouth

along the margins of the incision. Incision and drainage are not the treatments of choice, as they may reaccumulate fluid. Removal of the affected major salivary gland is required for recurrent and plunging ranulae.

Salivary Duct Cyst (Fig. 51.5) The **salivary duct cyst** is an epithelium-lined recess that arises within salivary gland ductal tissue, either the major or minor glands, as a result of an obstruction or developmental defect. They are slow-growing and asymptomatic fluctuant swellings that frequently develop in the lip, buccal mucosa, floor of mouth, or parotid gland. When superficial, they are blue or amber in color. Deep lesions do not alter the color of the mucosa. Treatment is excision.

Salivary Calculi (Sialoliths) (Fig. 51.6) **Sialoliths** or salivary stones (calculi) are calcified complexes within a salivary gland or duct that may obstruct salivary flow and cause floor-of-the-mouth swelling. They are usually round or oval and smooth or rough surfaced. Concentric laminations of different densities are often seen. Stones occur most frequently after age 25 years, twice as often in men as in women, and usually in the submandibular gland. The ascending course of the excretory duct, along with high mucous content and alkaline pH of the saliva, are significant factors in stone formation.

Obstruction of salivary flow by a sialolith in Wharton's duct results in a floor of the mouth swelling that is firm, tender, and painful. Acute symptoms often recur at mealtime. Swelling may extend along the course of the excretory duct and last for hours or days, depending on the blockage. The overlying mucosa usually remains pink, unless secondarily infected. In this case, the mucosa may turn red, and pus may emanate from the ductal opening. A sialolith can also cause a **salivary duct cyst** by increased intraductal pressure (see Fig. 51.5). Management involves appropriate occlusal radiographs, sialography (if no infection is present), and surgical removal of the sialolith. Localized cellulitis and fever require the use of antibiotics before invasive procedures.

Mucocoele (Mucus-Retention Phenomenon) (Figs. 51.7 and 51.8) The **mucocoele** is a soft fluctuant lesion involving the retention of mucus in subepithelial tissue, usually as a result of trauma (possibly iatrogenic) to a salivary gland duct. These clear or bluish swellings may occur on the lip (Fig. 49.1), floor of the mouth, ventral tongue, palate, or buccal mucosa. They are asymptomatic and <1 cm in diameter. The base of the lesion is usually sessile, although pedunculated bases are possible. Children and young adults are most frequently affected. Superficial lesions may heal spontaneously, whereas persistent lesions should be excised and examined microscopically. If the condition is managed properly, recurrences are rare.



Fig. 51.1. Dermoid cyst: below mylohyoid muscle.

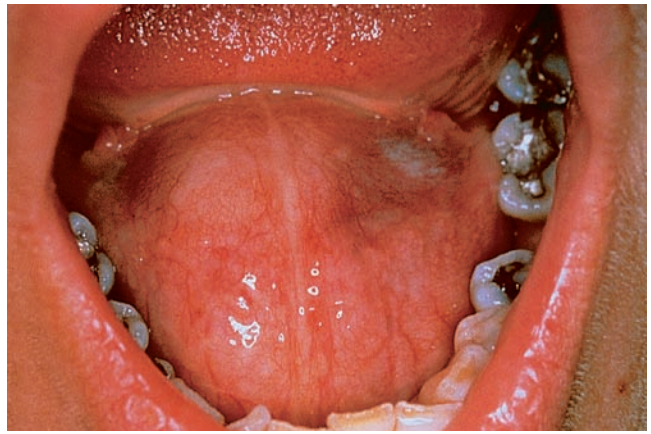


Fig. 51.2. Dermoid cyst: above mylohyoid muscle.

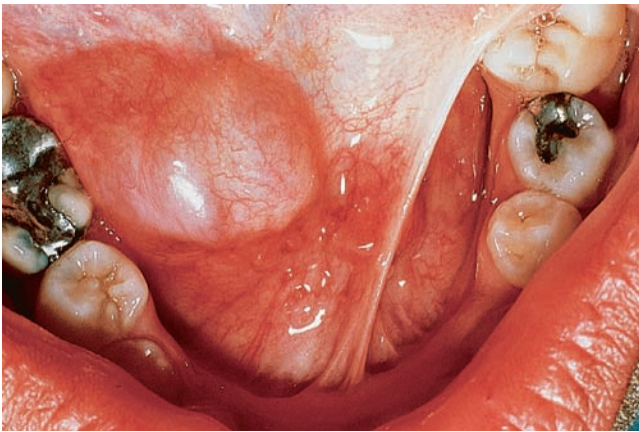


Fig. 51.3. Ranula: typical size, color, and translucency.



Fig. 51.4. Ranula: rare large lesion; impairs eating.



Fig. 51.5. Salivary duct cyst: caused by sialolith.



Fig. 51.6. Sialoliths: concentric laminations of several calculi.



Fig. 51.7. Mucocele: superficial translucent lesion.



Fig. 51.8. Mucocele: from trauma during crown preparation.

SWELLINGS OF THE PALATE

Palatal Torus (Torus Palatinus) (Fig. 52.1) A **palatal torus** is a bony exostosis (outgrowth) located in the midline of the hard palate posterior to the bicuspid teeth (Fig. 33.2). It affects approximately 20% of adults. They are frequently inherited, with a higher incidence in women than in men. After puberty, there is a tendency for slow growth.

Tori vary in size and shape. The flat torus sits on a broad base; the surface is smooth and minimally convex. The spindle torus is an enlarged, narrow, bony ridge along the midline of the palate. The lobular torus sits on a single base and is divided into lobules by one or several grooves. The covering mucosa is pale pink, thin, and delicate. The boundary of the lesion is distinct.

The palatal torus is frequently asymptomatic unless traumatized, with some patients unaware of the torus until after the traumatic episode. Palatal tori should be removed if they interfere with eating, speaking, playing a musical instrument, or a prosthetic appliance.

Lipoma (Fig. 52.2) The **lipoma** is a common benign mesenchymal tumor, but an uncommon intraoral finding. It is composed of adipocytes (fat cells) and appears as a well-circumscribed, dome-shaped yellowish submucosal mass. The buccal mucosa, tongue, floor of the mouth, and alveolar fold are common sites. The palate is a rare site of involvement. Treatment is excision (see Fig. 70.3).

Nasopalatine Duct Cyst (Incisive Canal Cyst) (Figs. 52.3 and 52.4) The **nasopalatine duct cyst** is a developmental cyst that arises from entrapped squamous or respiratory epithelial remnants of the nasopalatine duct within the incisive canal. It is the most common nonodontogenic cyst in the oral cavity. It may occur at any age and anywhere along the course of the incisive canal. However, the cyst is generally confined to palatal bone between the maxillary central incisors at the height of the incisive canal.

This cyst is usually asymptomatic and discovered as an incidental finding during routine examination. Symptomatic cysts are usually bacterially infected. Infrequently, the cyst of the incisive papilla arises entirely in soft tissue of the incisive papilla, where it appears as a small, superficial, fluctuant swelling. A mature incisive canal cyst may swell the entire anterior third of the palate. Radiographically, the cyst appears as a well-delineated, midline, symmetrically oval or heart-shaped radiolucency located between the roots of vital maxillary central incisors. The margin is sclerotic and contiguous with the incisive canal. Root divergence and root resorption of the central incisors are occasional findings associated with large lesions. A similar cyst that is located more posteriorly in the palate has been called the **median palatal cyst**. Current beliefs are that both cysts represent the same entity found in slightly different locations. All three conditions are treated by surgical enucleation.

Periapical Abscess (Figs. 52.5 and 52.6) A **periapical abscess** is a fluctuant, soft tissue swelling consisting of necrotic material and pus resulting from bacterial infection of

the pulp. It appears at the periapex of a diseased tooth that is often tender to percussion, mobile, and slightly high in occlusion. Regional lymphadenopathy, fever, malaise, and trismus are common accompanying features. Examination of the teeth and supporting tissues along with diagnostic testing reveal the offending non-vital tooth. Radiographs often show a periapical radiolucency.

Any abscessed maxillary tooth may produce a swelling of the palate. Generally, the swelling is red-purple, soft, tender, and lateral to the midline if a maxillary posterior tooth is involved. In contrast, an abscessed maxillary incisor may cause a midline swelling in the anterior third of the palate or lip. Aspiration or incision releases creamy yellow or yellow-green pus. Drainage, root canal therapy, or extraction is indicated to prevent spread of the infection. Antibiotics, analgesics, and antipyretic agents may also be needed.

Periodontal Abscess The differential diagnosis for a unilateral palatal swelling should include periodontal abscess—a localized infection involving the periodontium (refer to Figs. 37.7 and 37.8).

Lymphoid Hyperplasia (Fig. 52.7) **Lymphoid hyperplasia** is a rare, benign, reactive process that involves proliferation (rapid growth) of normal lymphoid tissue of the oropharynx (tonsils), tongue, floor of mouth, soft palate, or lymph node in response to an often unknown antigenic stimulus that is inhaled or ingested. Persons older than 30 years are most often affected. Clinical examination reveals a soft to firm swelling arising at the posterior extent of the hard palate that grows slowly (up to 3 cm), either unilaterally or bilaterally. The surface of the mature lesion is pink to purple, nonulcerated, and dome shaped or lumpy. Patients rarely report pain. Biopsy is recommended. However, the condition resolves spontaneously.

Lymphoma (Fig. 52.8) **Lymphoma** is a malignant neoplastic growth of lymphocytes. They are classified into **Hodgkin** or **non-Hodgkin lymphoma** and subdivided into nodal and extranodal disease. Epstein-Barr virus is often a factor in inducing malignant transformation of the lymphocytes. Non-Hodgkin lymphoma may develop at any lymphoid site, including cervical lymph nodes, mandible, palate, and, rarely, the gingiva. Primary lymphomas of the palate occur most commonly in patients older than 60 years but may be seen in younger patients, especially those with acquired immunodeficiency syndrome (AIDS). The tumor arises at the junction of the hard and soft palates as a soft, spongy, nontender, nonulcerated swelling. The surface is often lumpy and purplish. Rarely does it affect the underlying palatal bone. Early recognition and biopsy are important so that treatment may be initiated when the lesion is confined entirely to the palate. Irradiation is used to treat palatal lymphomas, whereas chemotherapy is used for disseminated disease.

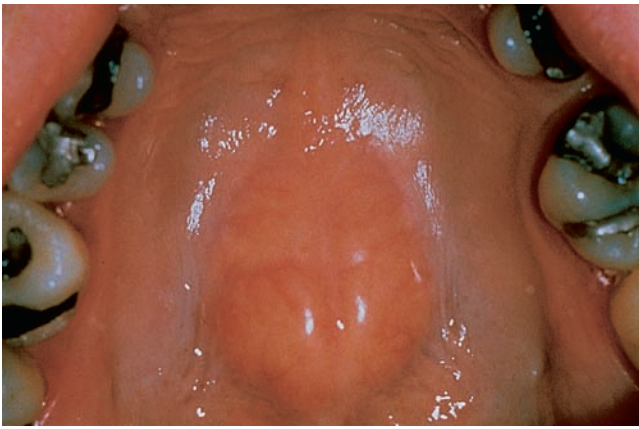


Fig. 52.1. Torus palatinus: flat, slight lobulation.

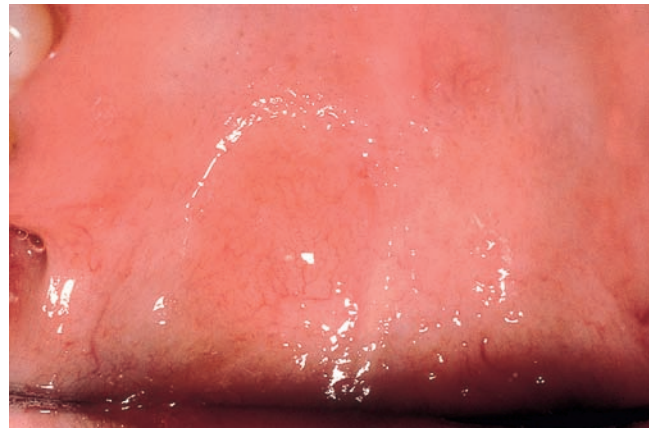


Fig. 52.2. Lipoma: palatal nodule; hypervascular surface.



Fig. 52.3. Nasopalatine duct cyst: anterior palate swelling.

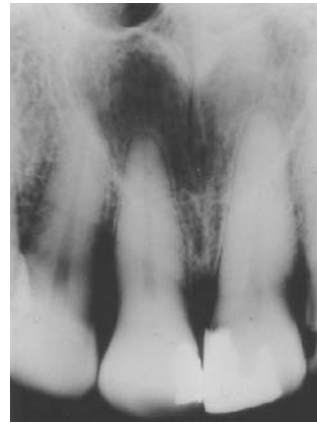


Fig. 52.4. Nasopalatine duct cyst: classic heart shape.



Fig. 52.5. Periapical abscess: from nonvital lateral incisor.



Fig. 52.6. Periapical abscess: large periapical radiolucency.

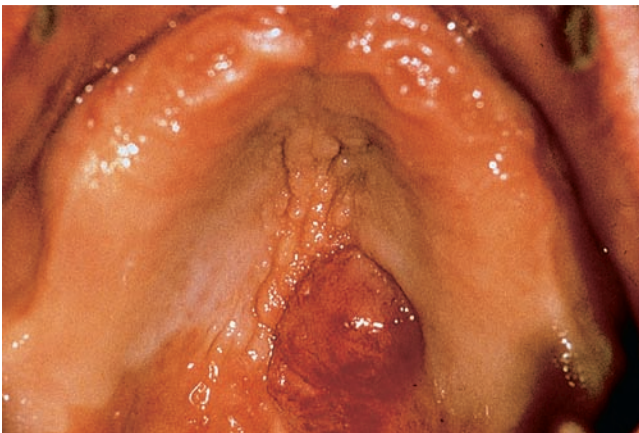


Fig. 52.7. Lymphoid hyperplasia: at posterior palate.



Fig. 52.8. Lymphoma of palate: nontender, with telangiectasia.

SWELLINGS OF THE PALATE: SALIVARY GLAND LESIONS

Sialadenitis (Fig. 53.1) **Sialadenitis** is inflammation of a salivary gland. It is often caused by obstruction and bacterial infection (bacterial sialadenitis) that arises when salivary flow is low because of dehydration or illness. This state allows pathogens (*Staphylococcus aureus*, *Streptococcus viridans*, and *Streptococcus pneumoniae*) to travel up the duct system and replicate. Viruses (mumps, coxsackie, echo) also can be causative. A distinct type of sialadenitis, **subacute sialadenitis**, affects the minor salivary glands of the palate of young men. It appears as a painful, reddish nodular swelling (up to 1 cm) that has an intact, nonulcerated surface. The cause is unknown, and biopsy is recommended.

Necrotizing Sialometaplasia (Figs. 53.2 and 53.3) **Necrotizing sialometaplasia** a benign, self-healing lesion of the accessory palatal salivary glands that is often confused clinically and histologically with carcinoma. This destructive inflammatory lesion begins after trauma and presents as a rapidly growing swelling on the lateral aspect of the hard palate, particularly in men older than 40 years. Tissue infarction as a result of vasoconstriction and ischemia caused by trauma or dental injection appears causative. Rarely, the soft palate or buccal mucosa is involved. Bilateral cases have been reported. The initial presentation is a small painless nodule that enlarges and causes pain. Within a few weeks, it ulcerates, and the pain diminishes. The size of the swelling varies, and growth to a diameter of 2 cm is possible. A deep central ulcer with a grayish pseudomembrane is characteristic. The ulcer is irregular and pebbly; the border is often rolled. Healing occurs spontaneously over 4 to 8 weeks or after a biopsy. Biopsy is recommended to rule out similar-appearing lesions, such as salivary gland tumors and malignant lymphoma. Histologically, it demonstrates squamous metaplasia of ductal epithelium, which may be misdiagnosed as mucoepidermoid carcinoma.

Benign Accessory Salivary Gland Neoplasm (Figs. 53.4 and 53.5) There are more than 10 types of benign salivary gland tumors. As many as 80% occur within the parotid gland, 10% in the submandibular gland, and about 10% to 20% in minor salivary glands. Most are pleomorphic adenoma, Warthin tumor, oncocytoma, or basal cell adenoma. Two of the more common types are discussed.

The **basal cell (monomorphic) adenoma** is a benign salivary gland tumor that can develop in the palate, most commonly in older women. It appears as a slow-growing dome-shaped mass. The tumor is encapsulated and consists of a regular glandular pattern and usually only one cell type. It lacks a mesenchymal component as seen in pleomorphic adenoma. Treatment is surgical excision.

The **pleomorphic adenoma** (benign mixed tumor) is the most common benign neoplasm of accessory salivary glands. It occurs more often in major than minor salivary glands. When the minor glands are affected, the palate is the most common location. Occurrences are

most frequent in women between the ages of 30 and 60 years. These neoplasms tend to occur lateral to the midline and distal to the anterior third of the hard palate.

The clinical presentation of the pleomorphic adenoma is a painless, firm, slow-growing, nonulcerated, dome-shaped swelling. Palpation reveals isolated softer areas and a smooth or lobulated surface. Slow persistent enlargement over a period of years is typical, and lesions may achieve sizes >1.5 cm in diameter. Histologically, it shows epithelial cells in a nestlike arrangement, with pools of myxoid, chondroid, and mucoid material. A distinct fibrous connective tissue capsule containing tumor cells surrounds and usually limits the extension of the tumor. Thorough excisional biopsy is recommended because the condition frequently recurs after simple enucleation or incomplete excision. Tumorous involvement of the capsule may play a role in recurrence.

Malignant Accessory Salivary Gland Neoplasm (Figs. 53.6-53.8)

There are more than 15 different types of malignant salivary gland tumors. **Adenoid cystic carcinoma** (cylindroma) and **mucoepidermoid carcinoma** are the two most common intraoral malignant neoplasms of the accessory salivary glands. Persons between the ages of 20 and 50 years are most frequently affected by mucoepidermoid carcinoma. The adenoid cystic carcinoma usually occurs after age 50 years. The adenoid cystic carcinoma occurs also in respiratory, gastrointestinal, and reproductive tissues, whereas mucoepidermoid carcinoma may occur in the skin, respiratory tract, or centrally within bone, particularly the mandible.

Malignant accessory salivary gland neoplasms occur frequently in the posterior palate. These neoplasms are usually asymptomatic, firm, dome-shaped swellings that occur lateral to the midline. The overlying tissue appears normal in the early stages, but the mucosa later becomes erythematous, with several small telangiectatic surface vessels. These neoplasms grow more quickly and are more painful than benign salivary gland tumors. Induration and eventual spontaneous ulceration are common. A bluish appearance or a mucous exudate emanating from the ulcerated tumor surface are distinctive features of mucoepidermoid carcinoma.

Treatment is radical excision. The prognosis varies, depending on the degree of histologic differentiation, the extent of the lesion, and the presence of metastasis. Adenoid cystic carcinoma rarely metastasizes but is an infiltrating malignancy with a propensity for distant spread by perineural invasion. Thus, lifetime follow-up is necessary. In contrast, the mucoepidermoid carcinoma infrequently metastasizes and is more easily cured by surgical means. Other malignant accessory salivary gland neoplasms include the acinic cell adenocarcinoma, polymorphous low-grade adenocarcinoma, carcinoma ex pleomorphic adenoma, carcinosarcoma, and malignant mixed tumor.



Fig. 53.1. Subacute sialadenitis: in young man.

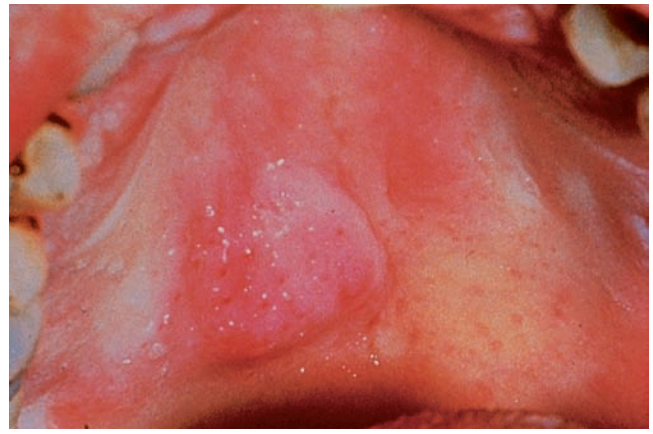


Fig. 53.2. Necrotizing sialometaplasia: painful swelling.



Fig. 53.3. Necrotizing sialometaplasia: ulcerative phase.

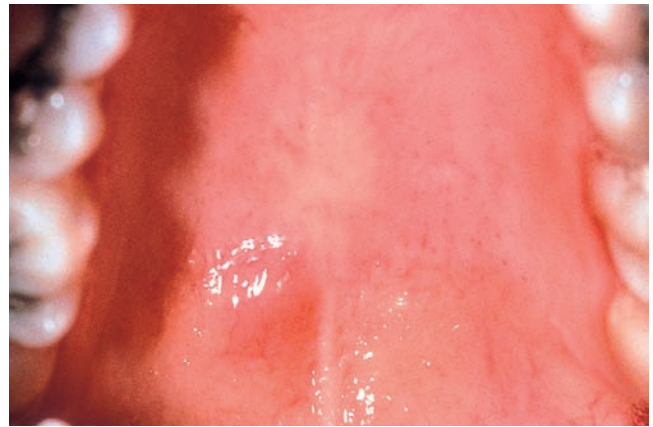


Fig. 53.4. Basal cell adenoma: painless nodule.



Fig. 53.5. Pleomorphic adenoma: strikingly firm nodule.

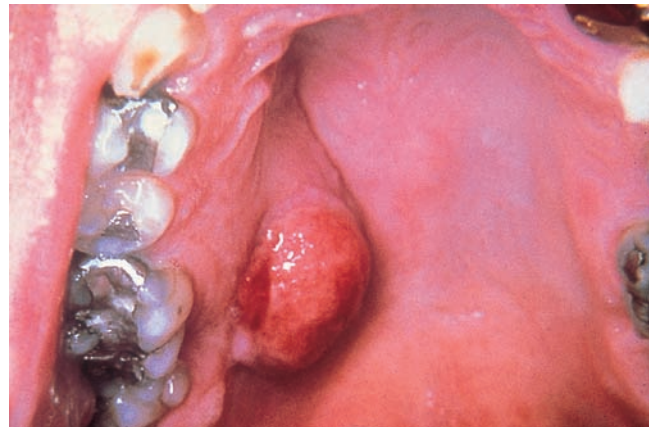


Fig. 53.6. Malignant mixed tumor: ulcerated.

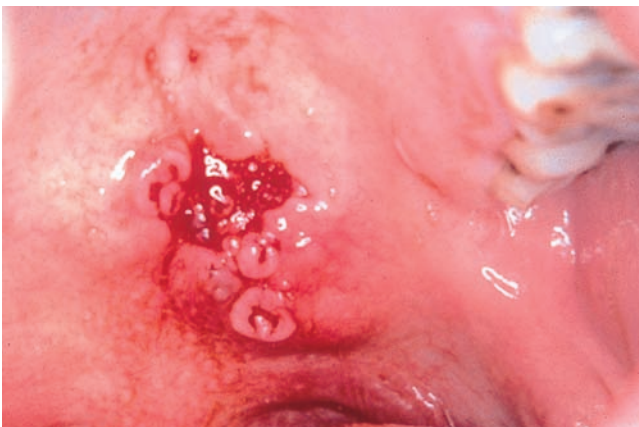


Fig. 53.7. Mucoepidermoid carcinoma: draining fistulas.



Fig. 53.8. Adenoid cystic carcinoma: surface ulceration.

SWELLINGS OF THE FACE

Odontogenic Infection (Figs. 54.1–54.8) Orofacial infections arise when microbes invade tissue, replicate, and overwhelm the local immune response. Infections are promoted by poor health and systemic disease, poor oral hygiene, and traumatic surgery. Oral infections are called odontogenic when they are tooth related. Most **odontogenic infections** arise as a consequence of pulpal necrosis, apical periodontitis, and pericoronitis. Almost all odontogenic infections are polymicrobial, consisting of anaerobic (65%) and aerobic (35%) bacteria. Obligate gram-negative anaerobes (*Prevotella*, *Fusobacterium*, *Bacteroides* species), anaerobic gram-positive organisms (*Peptostreptococcus* species), and facultative anaerobic gram-positive streptococci (*Streptococcus milleri*) are the most frequently identified organisms. Other contributors are *Lactobacillus*, *Diphtheroids*, *Actinomyces*, and *Eikenella* species. It is common for these organisms to be resistant to one or more frequently used antibiotics.

Odontogenic infections produce four classic features: **calor (heat)**, **dolor (pain)**, **rubor (redness)**, and **tumor (swelling)**. They begin as a soft tissue swelling adjacent to a tooth that slowly enlarges and produces dull pain and a bad taste. It may remain localized for a long time or progress to an abscess, parulis, cellulitis, or space infection. An **abscess** is an acute swelling that contains pus and necrotic debris. It appears as a well-localized swelling that is soft and occasionally pointed (Fig. 37.7). It is visible in or outside the mouth and drains spontaneously or when incised. The **parulis** is similar to the abscess, but the bacterial infection drains through a sinus tract onto the mucosal surface as a yellow-red papule (Fig. 41.1). In most cases, the parulis represents chronic infection and is asymptomatic. **Cellulitis** is an early feature of a spreading odontogenic infection; it represents a diffuse inflammatory response that has not yet localized. Cellulitis produces a diffuse, red, warm, hard swelling (generally over the cheek or mandible) that is firm and tender to palpation (Fig. 50.8). It may mature into an abscess and drain or become aggressive and spread. Spread of infection beyond anatomic boundaries through fascial planes (spaces) is called a **space infection**.

Buccal Space Infection (Figs. 54.1 and 54.2) A **buccal space infection** is most often caused by an odontogenic infection that has spread beyond the cortical plate, lateral to the buccinator muscle and anterior to the masseter muscle. It frequently arises from lateral migration of pathogenic bacteria that infect the periapex of a maxillary or mandibular molar. Entry into the space is usually gained at the posterior insertion of the buccinator muscle. Pain, swelling, fever, and chronic inflammation of a mandibular molar are the most prominent features.

Masseteric (Masticator) Space Infection (Figs. 54.3 and 54.4) A **masseteric space infection** is generally the result of an odontogenic infection that has spread posteriorly from

an infected mandibular molar or buccal space to a region between the masseter muscle and the lateral aspect of the ramus. The swelling is firm and nonfluctuant and overlies the angle of the mandible. Masseteric muscle involvement produces marked trismus (difficulty in opening). Computed tomography and magnetic resonance imaging are useful in defining this condition.

Infraorbital Space Infection (Figs. 54.5 and 54.6) Infection of the **infraorbital space** is the result of an infected maxillary tooth (usually a tooth anterior to the first molar) that has spread superiorly and involves the region lateral to the nasal ala and below the eye. Grave concern is raised when the infection encroaches on the eyelid or affects vision, because the ophthalmic (angular) veins lack valves and spread of infection to the brain is possible. **Cavernous sinus thrombosis** is a severe infection resulting from spread of infection via the angular veins to the brain.

Treatment of odontogenic infection involves four steps: (1) removal of the source of the infection, (2) establishment of drainage, (3) provision of antibiotics when needed, and (4) provision of supportive care. The infection is often controlled by instituting root canal therapy or extraction of the offending tooth when the infection is localized (for example, parulis, apical periodontitis). These treatments provide a pathway for drainage and reduce the bacterial load. Alternatively, incision and drainage can be performed to drain an abscess. If the condition is diffuse, as in cellulitis or pericoronitis, incision and drainage are generally unsuccessful and not recommended. Root canal therapy or extraction are used to treat cellulitis caused by a nonvital tooth, whereas pericoronitis is generally managed first with irrigation and antibiotics. When the pain of pericoronitis remits, extraction is performed. Guidelines for prescribing antibiotics include the presence of a spreading infection (regional lymphadenopathy, fever, swelling beyond anatomic boundaries) in combination with inability to establish drainage or immune compromise. Penicillin VK is the drug of choice, generally prescribed as 500 mg orally four times daily for 7 days.

Ludwig Angina (Figs. 54.7 and 54.8) **Ludwig angina** is a severe and rapidly spreading cellulitis that involves the submandibular, submental, and sublingual spaces bilaterally. These spaces are located between the tongue, hyoid bone, and lingual cortical plates of the mandible. It generally arises from an infected mandibular molar or a fractured (and infected) mandible. Spreading infection forces the tongue superiorly and posteriorly and forces submandibular tissues apically. This impinges on the airway and can result in airway obstruction. Aggressive use of antibiotics, culture, sensitivity, and multiple incisions and drains may be required to control the infection. In some patients, emergency procedures, such as a tracheostomy, are needed to sustain life.



Fig. 54.1. Buccal space infection: infected mandibular molar.

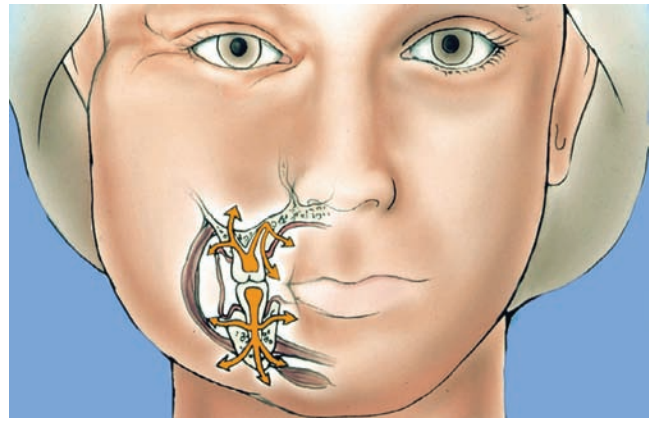


Fig. 54.2. Buccal space infection: anatomic illustration.



Fig. 54.3. Masseteric space infection: infected no. 19.

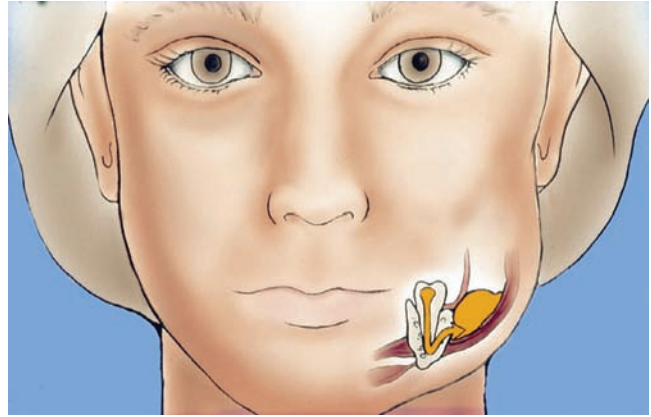


Fig. 54.4. Masseteric space infection: anatomic illustration.



Fig. 54.5. Infraorbital space infection: impinging on the eye.

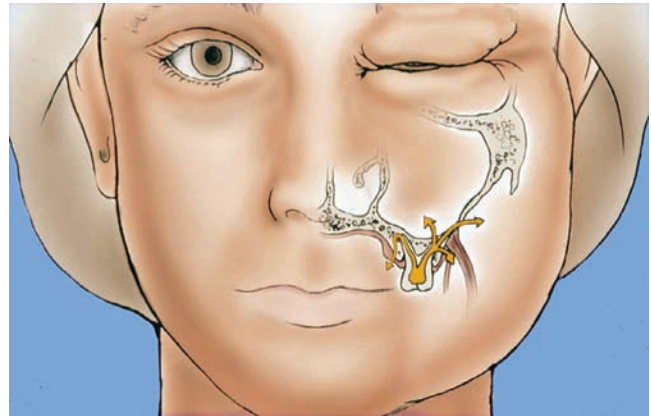


Fig. 54.6. Infraorbital space infection: anatomic illustration.



Fig. 54.7. Ludwig angina: requiring tracheostomy.

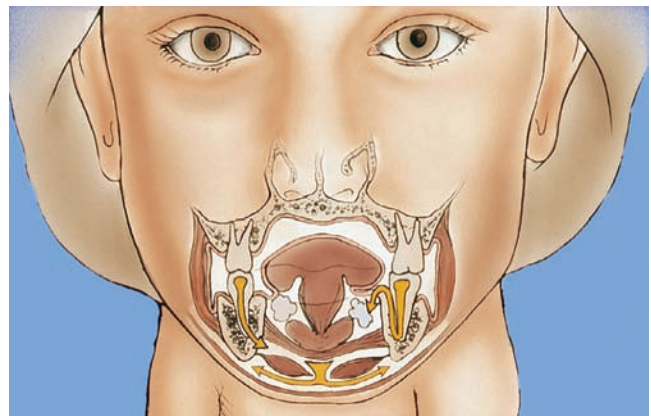


Fig. 54.8. Ludwig angina: anatomic illustration.

SWELLINGS OF THE FACE

Sialadenosis (Fig. 55.1) **Sialadenosis** is a painless noninflammatory swelling of major salivary glands. The condition indicates an underlying systemic disorder, such as alcoholism, anorexia nervosa, bulimia, diabetes, drug reaction, malnutrition, or HIV infection. It arises when acinar cells swell, probably because of dysregulation of autonomic innervation of the gland. Most cases begin as slow-growing, painless swellings of the parotid gland. It is often bilateral but can be unilateral. Salivary flow may be reduced. Microscopy shows hypertrophied acinar cells engorged with secretory granules and fat infiltration. Treatment attempts to control the systemic disease.

Warthin Tumor (Papillary Cystadenoma Lymphomatosum) (Fig. 55.2) **Warthin tumor** is a benign tumor that usually occurs after age 60 and almost exclusively in the parotid gland. It is thought to arise from entrapped glandular elements or proliferation of ductal epithelium. Smokers have eight times the risk as nonsmokers. There is a slight predilection for men. The tumors arise mostly in the tail of the parotid gland as a slow-growing, doughy, nodular mass. About 5% to 10% occur in parotid glands bilaterally. The submandibular and minor salivary glands are rarely affected. The tumor is composed of bilayered, oncocytic ductal epithelium that forms uniform rows and papillae surrounding cystic spaces. A lymphoid stroma surrounds the epithelium. Biopsy and excision are recommended. Recurrence and malignant transformation are rare.

Sjögren Syndrome (Fig. 55.3) **Sjögren syndrome** is an autoimmune disease in which the mouth and eyes become extremely dry. It is characterized by progressive lymphocytic infiltration that results in exocrine gland dysfunction. The cause of the disease remains unknown. It affects 1 in 2,000 persons, women in 90% of cases, and usually manifests between 35 and 50 years of age. The primary form, **sicca syndrome**, is limited to the salivary and lacrimal glands, producing dry eyes (xerophthalmia) and dry mouth (xerostomia). Oral and ocular disease accompanied by systemic disease (i.e., rheumatoid arthritis, systemic lupus erythematosus, and infiltrative disease of gastrointestinal and pulmonary exocrine glands) is known as **secondary Sjögren syndrome**. The syndrome develops slowly and involves progressive enlargement of the major salivary glands, particularly the parotid glands bilaterally. The glands become firm but not painful. Involvement of the pancreas or gall bladder results in abdominal symptoms. Labial salivary gland biopsy shows multiple lymphocyte aggregates adjacent to salivary acini. Serum markers, i.e., antinuclear antibodies (anti-SS-A and anti-SS-B) and rheumatoid factor, help confirm the diagnosis of secondary Sjögren syndrome. Management of dryness is attempted with artificial tears and saliva. Pilocarpine, cevimeline, fluoride, and chlorhexidine are useful. Patients are at increased risk for caries and the development of lymphoma.

Cushing Disease and Syndrome (Fig. 55.4) **Cushing disease** (hyperadrenocorticism) is a condition that results from

excess corticosteroid secretion from the adrenal gland. It is usually induced by a pituitary adenoma. **Cushing syndrome**, in contrast, results from chronic corticosteroid use. High serum cortisol levels cause fluid retention, hypertension, and hyperglycemia as well as truncal obesity, a round (moon-shaped) face with plethora (red), facial and truncal acne, a buffalo hump at the back of the neck, and purple abdominal striae. These features are caused by collagen wasting, dermal weakening, capillary fragility, and fluid accumulation. Treatment is directed toward eradicating tumors and correcting daily cortisol levels.

Masseter Hypertrophy (Fig. 55.5) **Masseter hypertrophy** is an increase in the size of the masseter muscle because of increased size of the cells. It occurs as a result of chronic muscle activity induced by tooth clenching, gum chewing, or bruxism. The masseter muscles are firm and nontender. Enlargement is most prominent over the angle of the mandible.

Neurofibromatosis (von Recklinghausen Disease) (Fig. 55.6) **Neurofibromatosis** is an inherited (autosomal dominant) disorder caused by a loss of a tumor suppressor gene (NF1 or NF2) that results in multiple neurofibromas (tumors involving nerve tissue) of the skin, mouth, bone, and gastrointestinal tract and skin pigmentations. The pigmentations affect the skin as café au lait spots and axilla as freckling (the Crowe sign) as well as the iris (Lisch nodules). The tumors appear after puberty as papules, nodules, or pendulous growths. In the mandible, they can expand the mandibular canal and cortical plates, produce facial swelling, or, rarely, undergo malignant transformation.

Cystic (Lymphangioma) Hygroma (Fig. 55.7) A **cystic hygroma** is a benign growth or hamartoma of lymphatic vessels (lymphangioma) that fail to drain lymph properly. As a result, lymph collects within the numerous dilated vessels. Most appear in the head, neck, and axilla as soft, compressible swellings. Airway obstruction may be a concern. Excision is generally successful.

Ewing Sarcoma (Fig. 55.8) **Ewing sarcoma** is a small, round cell malignancy caused by a chromosomal translocation (i.e., chromosome 11 switches with chromosome 12 or 22). The tumor appears to have a neuroectodermal origin. Persons younger than 25 years are most often affected. Most sarcomas arise in the femur, pelvis, and ribs; <5% arise in jaws. They are sometimes found in soft tissue without bony involvement. In the jaws, the mandible is most often involved. Patients present with swelling, pain, paresthesia, loose teeth, fever, leukocytosis, and an elevated sedimentation rate. The mass is soft when the tumor has penetrated the bony cortical plate. Radiographs show displaced teeth and a destructive radiolucent mass with ill-defined margins. Metastasis to lung, liver, and lymph nodes occurs frequently. Survival rates of 50% to 75% have been achieved with the combined use of radiation, surgery, and chemotherapy.



Fig. 55.1. Sialadenosis: of the parotid glands in a diabetic patient.



Fig. 55.2. Warthin tumor: bilateral parotid enlargement.



Fig. 55.3. Sjögren syndrome: dry eyes, parotid enlargement.

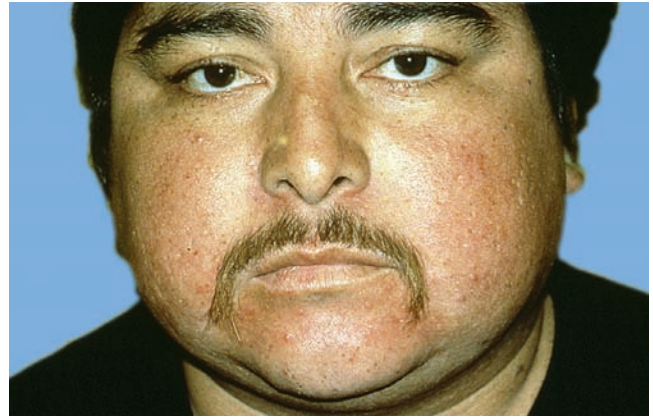


Fig. 55.4. Cushing syndrome: acne and red swollen face.



Fig. 55.5. Masseter hypertrophy: in a clench.



Fig. 55.6. Neurofibromatosis: asymmetrical enlargement.



Fig. 55.7. Cystic hygroma: soft, present since birth.



Fig. 55.8. Ewing sarcoma: rapidly growing malignancy.

CONDITIONS PECULIAR TO THE FACE

Angioedema (Fig. 56.1) **Angioedema** is a hypersensitivity reaction characterized by accumulation of fluid within facial tissues. The resulting tissues are soft, swollen, and itchy. It can be triggered by mechanical trauma, stress, infection, or an allergen. Most cases are acquired and result from IgE-mediated mast cell degranulation and release of histamine after exposure to an allergen (such as food) or physical contact. Histamine mediates capillary leakiness. Infections and autoimmune disease are less common triggers of the condition.

Angioedema produces facial swelling that develops within minutes or over a few hours. The swellings are usually uniform, diffuse, and symmetric. The lips are commonly affected, producing overly full, stretched, pliable swellings. Generally, the skin tone remains normal in color or is slightly red. The tongue, floor of the mouth, eyelids, face, and extremities may also be affected. Acquired angioedema is usually recurrent and self-limiting and poses little threat to the patient. Symptoms are limited to burning or itching. Management involves use of antihistamines, identification and withdrawal of allergenic stimuli, and stress reduction.

In the rare autosomal dominant **hereditary form**, angioedema is caused by an enzyme deficiency (type I) or a dysfunctional enzyme (type II) that results in activation of the complement pathway. Treatment involves avoidance of violent physical activity and trauma and prophylaxis with androgenic drugs, such as danocrine (Danazol). Angiotensin-converting enzyme (ACE) drugs used for high blood pressure and nonsteroidal anti-inflammatory drugs can also cause angioedema. ACE inhibitors can induce the condition by increasing bradykinin levels, not by the release of histamine. In this case, alternative drugs should be prescribed.

Emphysema (Fig. 56.2) **Emphysema** is defined as the abnormal presence of air in tissue. Although rare in dentistry, it most often occurs during a surgical procedure when compressed air from an air-driven handpiece or syringe is forced under a mucoperiosteal flap, through an open pulp chamber, or intra-alveolarly. The air becomes entrapped in subcutaneous tissue or a fascial plane. Under these circumstances, the soft tissues adjacent to the surgical site distend within minutes of the procedure. The swelling is soft or mildly firm and yields a distinctive crackling sound (crepitus) on palpation. The entrapped air can migrate along fascial planes through the neck to the sternum, into the prevertebral fascia and mediastinum, toward the temporal and orbital regions, or into the vascular system. Emphysema is associated with risks for infection, air embolism, and death. Broad-spectrum antibiotics are recommended to prevent infection. Signs suggestive of embolic sequelae, such as sudden vision

changes, dyspnea, altered heart rate, or loss of consciousness, warrant immediate emergency care.

Postoperative Bleeding (Figs. 56.3–56.6) Facial swelling can result from orofacial bleeding when there is excess bleeding or blood that lacks an escape route through the epithelium. The end result is **purpura**, the accumulation of blood within the subcutaneous or submucosal tissues. Purpura is classified according to the size of the lesion produced. **Petechiae** are pinpoint, flat, round red spots under the skin surface caused by hemorrhage from the capillaries (bleeding into the skin). A small bruise, larger than a petechia, caused by blood leaking from ruptured blood vessels into the surrounding tissues is called an **ecchymosis**. A **hematoma** is a large localized collection of blood in a tissue, organ, or body space resulting from a broken blood vessel.

In dentistry, hematomas are commonly caused by the inadvertent needle penetration into the posterior superior alveolar vein. Swelling occurs without pain within seconds or minutes of the trauma. The swelling grows uniformly and without color change in the posterior cheek region, over and in front of the mandibular ramus. The hematoma is slightly firm and compressible. It may cause tissue tenderness, nerve paresthesia, and muscle trismus. Pressure and ice should be applied to the hematoma during the first 24 hours. Thereafter, heat should be applied to dissipate the blood. Depending on its size, the lesion may take more than 1 week to resolve and may discolor the skin. During the healing phase, the skin color fades from purple to brown, then tan and yellow. Antibiotics should be considered if the hematoma is caused by severance of a vessel with a contaminated instrument.

Bell Palsy (Figs. 56.7 and 56.8) **Bell palsy** is a sudden weakness and paralysis of cranial nerve VII on one side of the face. The condition results in the inability to move the muscles of facial expression on the affected side. Events that damage or traumatize the facial nerve are most frequently causative and include surgery, tooth extraction, infection, or exposure to cold. About one third of cases are linked to reactivation of herpesvirus within the geniculate ganglion. All persons are susceptible, but the condition is seen most often in middle-aged adults (slightly more often in women). The palsy presents with abrupt onset of inability to raise the forehead skin, close the eye, and lift the corner of the mouth of the affected side. Eye watering (crocodile tears), mouth drooling, and loss of taste are common consequences. Without treatment, the palsy may be temporary or permanent. Antiviral (valacyclovir) and prednisone therapy has proven to be effective in treating Bell palsy. Palliative care to protect the eye from corneal ulceration should also be provided.



Fig. 56.1. Angioedema: caused by contact with latex rubber.

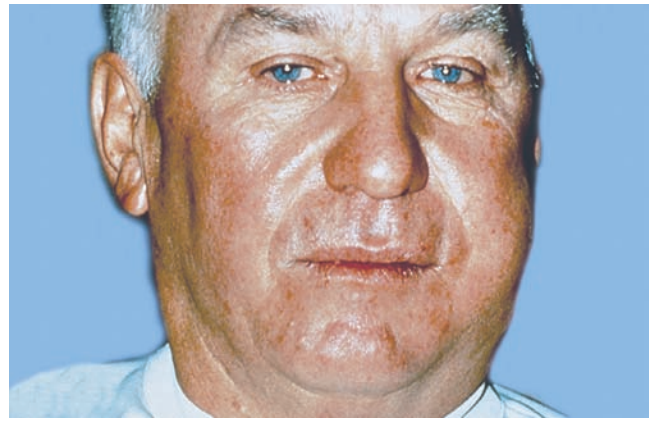


Fig. 56.2. Air emphysema: after flap surgery.



Fig. 56.3. Hematoma: posterior alveolar vein injured.*



Fig. 56.4. Hematoma: 1 week later.*



Fig. 56.5. Surgical trauma: swelling 2 days post-oral surgery.†



Fig. 56.6. Healing of postsurgical swelling: 2 weeks later.†

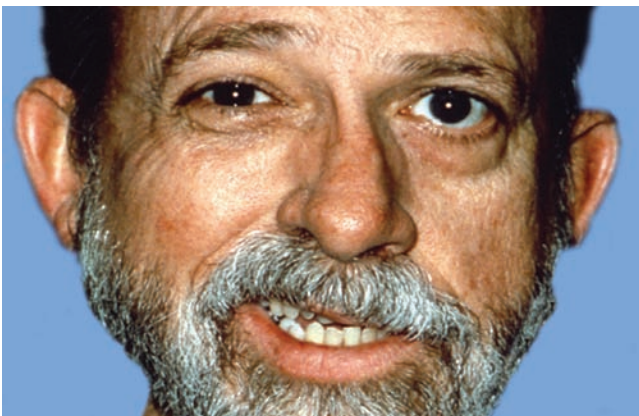


Fig. 56.7. Bell palsy: inability to lift left side of face.

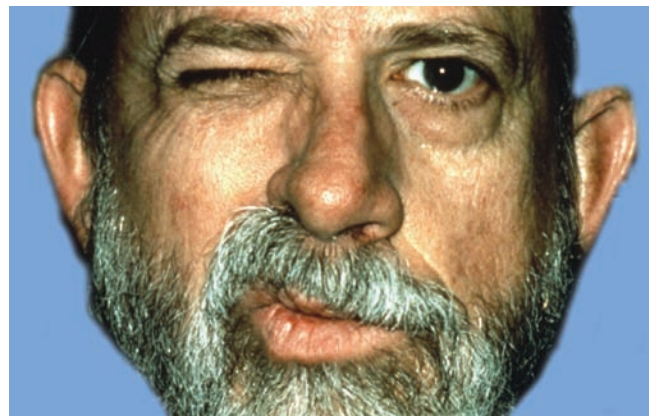


Fig. 56.8. Bell palsy: inability to close left eyelids.

CASE STUDIES

CASE 15. (Fig. 56.9) This 79-year-old Caucasian man was visited in a nursing home. His oral care is dependent largely on the nursing staff. During your oral exam, you notice this growth. The nursing staff believe that it was not present a few days ago.



1. Describe the clinical findings.
2. What is the fluid on the surface of the lesion?
3. Is this a normal finding, a variant of normal, or disease?
4. Do you expect this condition to be symptomatic?
5. List conditions you should consider in the differential diagnosis, and note which condition is most likely the correct diagnosis.
6. Why is this lesion pink and not clear or bluish in color?
7. What do you notice about the teeth and periodontium?
8. Does the presence of this soft tissue lesion prevent the provision of periodontal debridement?

CASE 16. (Fig. 56.10) This 29-year old man comes to your dental office for routine dental work. He has no dental symptoms and does not have a fever. The lesion does not rub off. He claims to have multiple sexual partners.



1. Describe the clinical findings. Include normal and abnormal findings.
2. Do you expect this condition to be symptomatic?
3. Is this a normal finding, a variant of normal, disorder, or disease?
4. What conditions should you consider in the differential diagnosis?
5. This condition is more common in:
 - A. HIV-seronegative men
 - B. HIV-seropositive men
 - C. HIV-seropositive women
 - D. HIV-seronegative women
6. True or false: This condition can occur in persons who take immunosuppressive drugs for organ transplantation.
7. True or false: This condition is limited to the tongue.
8. Why is it significant that the lesion can not be rubbed off?

Intraoral Findings by Color Changes

Dental Objectives:

- Define and use diagnostic terms that describe white, red, and pigmented lesions of oral mucosa.
- Recognize the causes and clinical features of white, red, and pigmented lesions of oral mucosa.
- Use the diagnostic process to distinguish similar-appearing white, red, and pigmented lesions of oral mucosa.
- Recommend appropriate treatment options for white, red, and pigmented lesions of oral mucosa.

Dental Hygiene Objectives:

- Define and use diagnostic terms that describe white, red, and pigmented lesions of oral mucosa.
- In the clinical setting, document the characteristics of white, red, and pigmented lesions of the oral mucosa in terms of:
 - a. Anatomic location
 - b. Color
 - c. Border
 - d. Configuration
 - e. Type
- Identify conditions discussed in this section that (1) require the attention of the dentist and/or (2) affect the delivery of periodontal debridement and oral hygiene measures.

In the figure legends, *[†] denotes the same patient.

WHITE LESIONS

Fordyce Granules (Figs. 57.1 and 57.2) **Fordyce granules** are sebaceous glands found within the oral mucosa. Technically speaking, they are sebaceous **choristomas** (i.e., normal tissue in an abnormal location); their normal location is within the upper layer of the dermis (skin). These asymptomatic granules consist of individual sebaceous glands that are 1 to 2 mm in diameter. Characteristically, they appear as white, creamy white, or yellow slightly raised papules on the buccal mucosa and vermillion of the upper lip. They usually occur in multiples, forming clusters, plaques, or patches. Enlarged clusters may feel rough to palpation and to the patient's tongue. They are sometimes an isolated finding. Less common locations include the labial mucosa, retromolar pad, attached gingiva, tongue, and frenum.

Fordyce granules arise from sebaceous glands embryologically entrapped during fusion of the maxillary and mandibular processes. They become more apparent after sexual maturity as the sebaceous system develops. Rarely, an intraoral hair may be seen in association with the condition.

Fordyce granules occur in approximately 80% of adults, and no race or sex predilection has been reported. However, the density of granules per mucosal area is greater in men than women. Histologic examination shows rounded nests of clear cells, 10 to 30 per nest, in the lamina propria and submucosa. These cells have darkly staining, small, centrally located nuclei. The clinical appearance is adequate for diagnosis of Fordyce granules. Biopsy is not usually required.

Linea Alba (Figs. 57.3 and 57.4) The **linea alba** ("white line") is a common intraoral finding that appears as a raised white wavy line of variable length and prominence located at the level of the occlusion on the buccal mucosa. Generally, this asymptomatic white line is 1 to 2 mm wide and extends horizontally from the second molar to the canine region of the buccal mucosa, ending at the caliculus angularis (Fig. 1.3). The lesion is most often found bilaterally and cannot be rubbed off. It develops in response to frictional activity of the teeth, which results in thickened (hyperkeratotic) epithelial changes. The condition is often associated with crenated tongue and may be a sign of pressure, bruxism, clenching, or sucking trauma. The clinical appearance is diagnostic. No treatment is required.

Leukoedema (Figs. 57.5 and 57.6) **Leukoedema** is an opalescent, milky-white, or gray surface change of the buccal mucosa. This common mucosal variant is associated with dark-pigmented persons but may be seen infrequently in lighter-pigmented persons. The incidence of leukoedema tends to increase with age, and 50% of

African American children and 92% of African American adults are affected. The labial mucosa, soft palate, and floor of the mouth are less common locations of occurrence.

Leukoedema is usually faint and bilateral. Close examination of leukoedema reveals fine white lines and wrinkles. Exaggerated and long-standing cases have overlapping folds of tissue. Prominence of the lesion is related to the degree of underlying melanin pigmentation, level of oral hygiene, and amount of smoking. The borders of the lesion are wavy and diffuse; they fade into adjacent tissue, which makes it difficult to determine where the lesion begins and ends. The condition is diagnosed by stretching the mucosa, which causes the white appearance to significantly diminish or disappear in some cases. Wiping the lesion fails to remove it. The cause of leukoedema is unknown, although it is more severe in smokers and diminishes with smoking cessation. Histologic examination of biopsy specimens shows increased epithelial thickness with prominent intracellular edema of the spinous (middle) layer without evidence of inflammation. No serious complications are associated with this lesion, and no treatment is required.

Morsicatio Buccarum (Figs. 57.7 and 57.8) **Morsicatio buccarum**, derived from the latin word *morsus* (bite), is the term used to describe the changes in oral mucosa that are due to cheek biting or cheek chewing. Cheek biting or chewing is a common nervous habit that produces a progression of mucosal changes. Initially, slightly raised irregular white plaques appear in a diffuse pattern that cover areas of trauma. Increased injury produces a hyperplastic response that increases the size of the plaque. A linear or striated pattern is sometimes observed that contains thick corrugated areas and intervening zones of erythema. Persistent injury leads to an enlarging plaque with irregular zones of traumatic erythema and ulceration.

Chewing of the oral mucosa is usually seen on anterior buccal mucosa and less frequently on the labial mucosa. The lesions may be unilateral or bilateral and can occur at any age. No sex or race predilection has been reported. Diagnosis requires visual or verbal confirmation of the nervous habit. Although morsicatio buccarum has no malignant potential, patients should be advised of the mucosal alterations. Because of the similar clinical appearance, speckled leukoplakia and candidiasis should be ruled out. Microscopic examination of biopsied tissue shows a normal maturing epithelial surface with a corrugated and thickened parakeratotic surface and minor subepithelial inflammation.

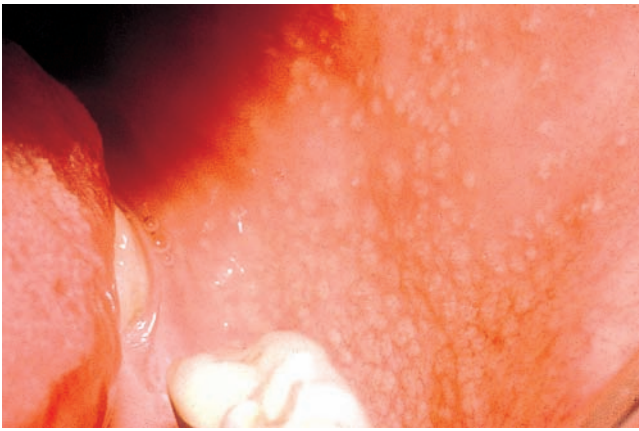


Fig. 57.1. Fordyce granules: clustered in buccal mucosa.

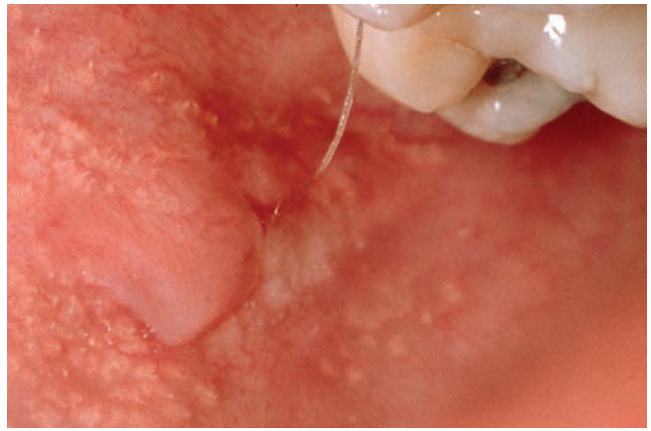


Fig. 57.2. Fordyce granules: with rare intraoral hair.



Fig. 57.3. Linea alba: on buccal mucosa.



Fig. 57.4. Prominent linea alba.



Fig. 57.5. Leukoedema: of buccal mucosa.



Fig. 57.6. Leukoedema: prominent in a smoker.



Fig. 57.7. Morsicatio buccarum: caused by cheek biting.



Fig. 57.8. Morsicatio buccarum: of labial mucosa.

WHITE LESIONS

White Sponge Nevus (Familial White Folded Dysplasia) (Figs. 58.1 and 58.2) **White sponge nevus** is an inherited condition characterized by the appearance of asymptomatic, white, folded, spongy plaques. The lesions often exhibit a symmetric wavy pattern. This condition is a relatively rare genodermatosis caused by point mutations in genes that regulate keratin 4 and keratin 13 production. As a result, epithelial maturation and exfoliation are altered. It appears at birth or early childhood, persists throughout life, and exhibits no race or sex predilection. Because it has an autosomal dominant pattern of transmission, several family members are typically affected.

The most common location of white sponge nevus is the buccal mucosa bilaterally, followed by labial mucosa, alveolar ridge, and floor of the mouth. It may involve the entire oral mucosa or be distributed unilaterally as discrete white patches. The gingival margin and dorsal tongue are almost never affected, although the soft palate and ventrolateral tongue are commonly involved. The size of the lesions vary. Extraoral mucosal sites may involve the nasal cavity, esophagus, larynx, vagina, and rectum. The oral features are remarkably similar to those of **hereditary benign intraepithelial dyskeratosis**. However, eye involvement occurs with the latter. Microscopically, white sponge nevus shows prominent parakeratosis, thickening and clearing of the spinous layer, and perinuclear tangles of keratin tonofilaments. No treatment is required.

Traumatic White Lesions (Figs. 58.3–58.6) **Traumatic white lesions** are caused by many physical and chemical irritants, such as frictional trauma, heat, topical use of aspirin, excessive use of mouthwash, caustic liquids, and even toothpastes. Frictional trauma is often noted on the attached gingiva. It is caused by excessive toothbrushing, movement of oral prostheses, and chewing on the edentulous ridge. With time, the mucosa becomes thickened and develops a roughened white surface that does not wipe off. Pain is absent. Histologic examination reveals hyperkeratosis.

Acute trauma can produce a white peeling or corrugated lesion if superficial layers of mucosa are damaged. Lesions usually appear as white patches with diffuse irregular borders. Underneath is a raw, red, or bleeding surface. Moveable mucosa is more susceptible to trauma than is attached mucosa. Pain relief and healing occur within days of removing the cause.

Another white lesion caused by trauma is a **scar**. It represents a fibrous healing response of the dermis. Scars are often asymptomatic, linear, whitish pink, and sharply delineated. A thorough history may reveal previous injury, recurrent ulcerative disease, seizure disorder, or previous surgery.

Leukoplakia (Figs. 58.7 and 58.8) **Leukoplakia** is a clinical term for a white plaque or patch that cannot be rubbed off and cannot be classified clinically as any other disease. Persons of any age may be affected, but the majority of cases occur in men between the ages of 45 and 65 years.

Leukoplakias are protective reactions against chronic irritants. Tobacco, alcohol, syphilis, vitamin deficiency, galvanism, chronic friction, ultraviolet radiation, and candidiasis have been implicated in causing these lesions. Leukoplakias vary considerably in size, location, and clinical appearance. Preferential sites are the lateral and ventral tongue, floor of the mouth, alveolar mucosa, lip, soft palate–retromolar trigone, and mandibular attached gingiva. The surface may appear smooth and homogeneous, thin and friable, fissured, corrugated, verrucoid, nodular, or speckled. Lesions can vary in color, from faintly translucent white, gray, or brown-white.

A classification system offered by the World Health Organization recommends two divisions for oral leukoplakias: **homogeneous** and **nonhomogeneous**. Nonhomogeneous leukoplakias are subdivided into **erythroleukoplakia** (white lesion with large red component), **nodular** (white lesion with raised and pebbly surface), **speckled** (white lesion with small red components), and **verrucoid** (white lesion with raised corrugated surface).

Most leukoplakias (80%) are benign; the rest are dysplastic (premalignant) or cancerous. The clinical challenge lies in determining which are premalignant or malignant, because 1% to 4% of leukoplakias progress to carcinoma within 20 years. High-risk sites of malignant transformation include the floor of the mouth, lateral and ventral tongue, uvulopalatal complex, and lips.

Proliferative verrucous leukoplakia is a persistent leukoplakia with a verrucoid (wartlike) appearance and a high risk for malignant transformation (Fig. 58.8). It is unusual leukoplakia because it has a strong female predilection, an infrequent association with smoking, and an occasional association with human papillomavirus infection. It occurs at any adult age but most commonly after age 60. Lesions are multifocal and have a white, corrugated or pebbly exophytic surface (pebbly outgrowths or wartlike outgrowths). Lesions spread slowly, rarely regress, and tend to recur after excision. About 70% of these leukoplakias developed into carcinoma.

Leukoplakias with localized red areas confer a high risk for carcinoma. Evidence indicates that nonhomogeneous leukoplakias, particularly oral **speckled leukoplakias**, represent epithelial dysplasia in about half of the cases and have the highest rate of malignant transformation among intraoral leukoplakias. *Candida albicans*, often associated with oral speckled leukoplakias, may have a role in the dysplastic changes seen.

The initial step in the treatment of leukoplakia is to eliminate any irritating and causative factors, then observe for healing. Unexplained persistent oral leukoplakias must be biopsied. Several biopsy sites may be necessary for diffuse lesions. Nonhomogeneous or reddish areas of the lesion should always be selected for biopsy because they are associated with a higher risk for dysplasia and malignant transformation.

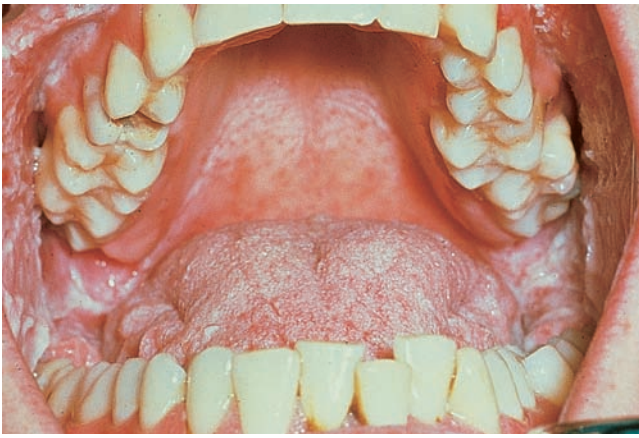


Fig. 58.1. White sponge nevus: buccal mucosa, soft palate.*



Fig. 58.2. White sponge nevus: of buccal mucosa.*



Fig. 58.3. Traumatic white lesion: toothbrushing associated.



Fig. 58.4. Traumatic white lesion: frictional keratosis.



Fig. 58.5. Traumatic white lesion: burn from topical aspirin.



Fig. 58.6. Leukoplakia: hyperkeratosis of soft palate.

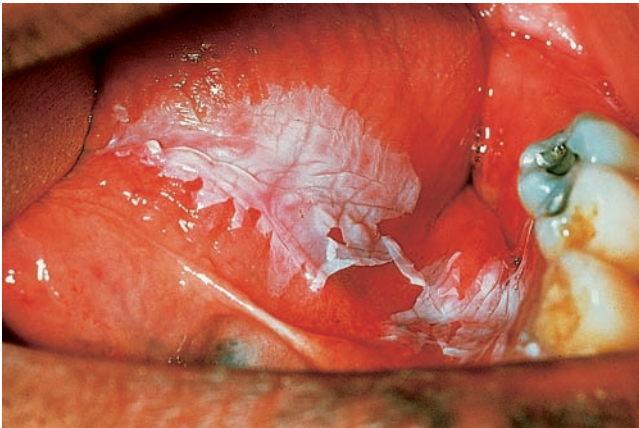


Fig. 58.7. Leukoplakia: of floor of mouth and ventral tongue.



Fig. 58.8. Proliferative verrucous leukoplakia: spreading.

TOBACCO-ASSOCIATED WHITE LESIONS

Cigarette Keratosis (Figs. 59.1 and 59.2) Keratosis is a condition characterized by thickened patches on the skin. **Cigarette keratosis** is a specific reaction evident in persons who smoke nonfiltered or marijuana cigarettes to a very short length. The lesions, which approximate each other on lip closure, involve the upper and lower lips at the location of cigarette placement. These keratotic patches are about 7 mm in diameter and are located invariably lateral to the midline. Raised white papules are evident throughout the patch, producing a roughened texture and firmness to palpation. Cigarette keratoses may extend onto the labial mucosa, but the vermilion border is rarely involved. Elderly men are most commonly affected. Smoking cessation usually brings about resolution. The development of ulcer and crust formation should raise the suspicion of neoplastic transformation.

Nicotine Stomatitis (Smoker's Palate) (Figs. 59.3 and 59.4) **Nicotine stomatitis** is a direct response of oral mucosa to prolonged pipe and cigar smoking. Its severity is correlated with the intensity and duration of smoke exposure. It is usually found in middle-aged and elderly men, in unprotected palatal regions (not covered by a maxillary denture) that contain minor salivary glands, that is, posterior to the palatal rugae, on the soft palate and sometimes extending onto the buccal mucosa. Rarely, the dorsum of the tongue is affected. These tobacco-associated changes of the tongue have been termed **glossitis stomatitis nicotina**.

Nicotine stomatitis shows progressive changes with time. The irritation initially causes the palate to become diffusely erythematous. The palate eventually becomes grayish white secondary to hyperkeratosis. Multiple discrete keratotic papules with depressed red centers develop that correspond to dilated and inflamed excretory duct openings of the minor salivary glands. The papules enlarge as the irritation persists but fail to coalesce, producing a characteristic cobblestone (par-boiled) appearance of the palate. Isolated but prominent red-centered papules are common. Brown staining of the lingual surface of the posterior teeth is commonly associated with this condition. Whether nicotine stomatitis arises as a consequence of heat or tobacco is a matter of debate. However, the lack of malignant progression suggests that the heat may be more contributory to the lesion than the chemicals in tobacco smoke. Reverse cigarette smoking (lit end placed in the mouth—common to persons from India) produces similar findings. Smoking cessation usually results in regression. Biopsy is rarely needed to confirm the diagnosis.

Snuff Dipper's Patch (Tobacco Chewer's Lesion, Snuff Keratosis) (Figs. 59.5 and 59.6) A wrinkled yellow-white area in the mucobuccal fold—or mandibular buccal or labial mucosa—suggests persistent intraoral use of unburned tobacco. The hard palate, floor of the mouth, and ventral tongue may also be affected if tobacco is placed in the maxillary vestibule or beneath the tongue. Smokeless

tobacco is marketed in various forms (snuff, dip, plug, or quid) and leaves its characteristic mark at the preferential site of tobacco placement. Posterior sites are commonly used for dip, plug, or quid, whereas anterior sites are preferred for snuff. Persons who vary placement of the tobacco have multiple, less prominent oral lesions. Male teenagers are most frequently affected, largely because of intensive marketing and peer and sports associations. Higher prevalence is found in southern and Appalachian states.

Early **snuff dipper's patches** are pale pink keratoses with corrugated or wrinkled surfaces. The color may progress from white and yellow-white to yellow-brown as hyperkeratosis and exogenous staining occur. Lesions are asymptomatic and often at least 1 cm in diameter.

Long-term use of smokeless tobacco is associated with periodontal recession, caries, epidermal dysplastic changes, and carcinoma. The dysplastic changes, which take many years to develop, are associated with the carcinogenic nitrosamines present in the tobacco. To achieve resolution, cessation of use is recommended. If normal appearance does not return 14 days after cessation, biopsy is necessary.

Verrucous Carcinoma (of Ackerman) (Figs. 59.7 and 59.8) **Verrucous carcinoma** appears as a warty, exophytic, white-and-red mass that is firm to palpation. Some would describe it as cauliflowerlike or papulonodular tumor. It is a low-grade, nonmetastasizing, variant malignant squamous cell carcinoma and is 25 times less common than squamous cell carcinoma. It most frequently arises in association with long-term tobacco use, especially smokeless tobacco at the site of chronic placement. About 30% of cases are associated with human papillomavirus infection.

The buccal mucosa, vestibule, mandibular gingiva, and palate are the most common oral sites of verrucous carcinoma. Men older than 60 years who have used smokeless tobacco for many years are most often affected. The disease is rare in persons younger than 40 years and in persons who do not use tobacco. Nonoral sites can also be affected.

Verrucous carcinoma has a distinctive surface appearance. A characteristic feature is a white-gray, undulating keratotic surface with pink-red pebbly papules throughout. Lateral growth, which usually exceeds vertical growth, leads to an increase in mass and diameters of several centimeters. Large lesions can be locally destructive by invading and eroding the underlying alveolar bone. Similar-appearing lesions include verrucous epithelial hyperplasia, pyostomatitis vegetans, and proliferative verrucous leukoplakia.

Recommended treatment for verrucous carcinoma is wide surgical excision. After surgery, the long-term prognosis of affected patients is better than squamous cell carcinoma and improves when the use of smokeless tobacco is discontinued.

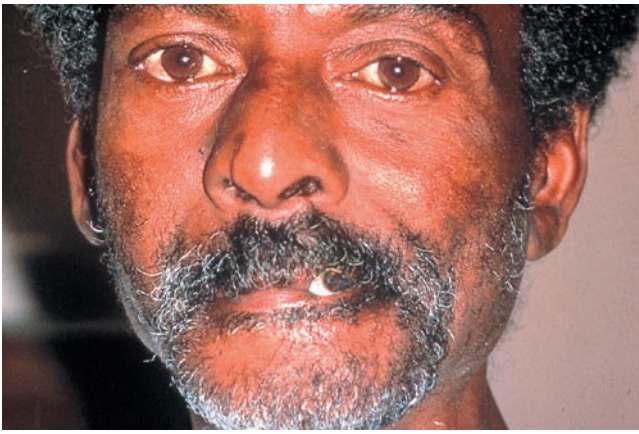


Fig. 59.1. Cigarette keratosis: user of unfiltered cigarettes.*



Fig. 59.2. Cigarette keratosis: on labial mucosa.*

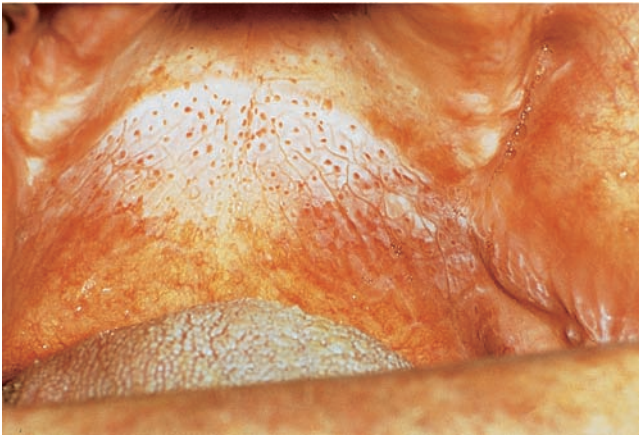


Fig. 59.3. Nicotine stomatitis: prominent on soft palate.



Fig. 59.4. Nicotine stomatitis: in a reverse smoker.



Fig. 59.5. Snuff dipper's patch: under chewing tobacco.



Fig. 59.6. Snuff dipper's patch: of mucobuccal fold.

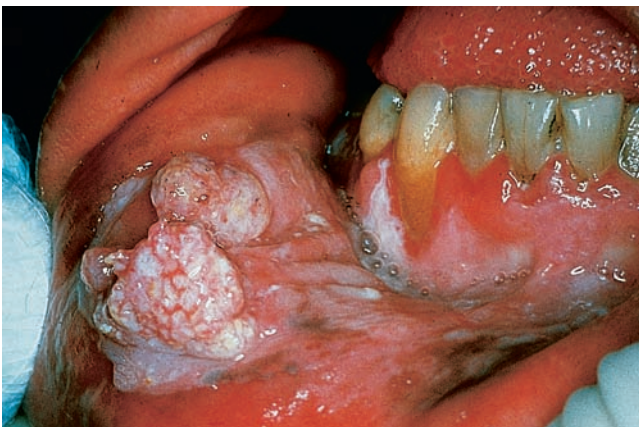


Fig. 59.7. Verrucous carcinoma: of the labial mucosa.

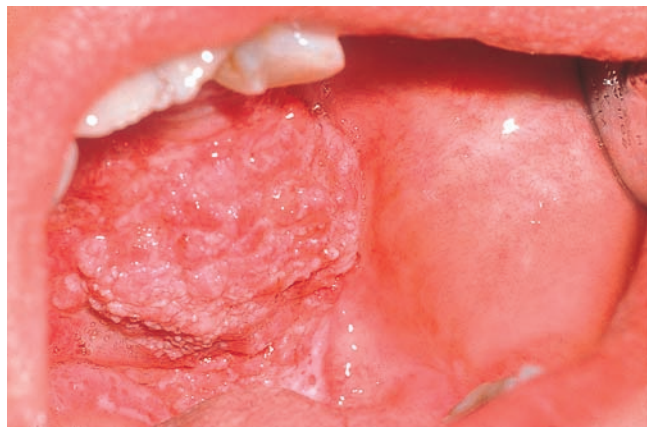


Fig. 59.8. Verrucous carcinoma: maxillary alveolar ridge.

RED LESIONS

Many lesions appear red because they have a vascular component. This section discusses red lesions associated with abnormalities of blood vessels and blood.

Purpura (Petechiae, Ecchymoses, Hematoma) (Figs. 60.1–60.4)

Purpura are demarcated discolorations produced by bleeding underneath the surface of the skin. They represent a sign either of trauma (iatrogenic, factitial, or accidental) or an underlying abnormality, such as a platelet or clotting disorder, capillary fragility, or infection. Purpura initially appears bright red but tends to discolor with time, becoming purplish blue and later brown-yellow. Because these lesions consist of extravasated blood, they do not blanch on diascopy (pressure with a glass slide).

There are three types of purpura—petechiae, ecchymoses, and hematoma—classified by size. **Petechiae** are pinpoint, nonraised, circular red spots. The soft palate is the most common intraoral location for multifocal petechiae. Palatal petechiae may represent an early sign of viral infection (infectious mononucleosis), scarlet fever, leukemia, platelet disorders, or coagulation disorders. They may also indicate rupture of palatal capillaries caused by coughing, sneezing, vomiting, or fellatio. Suction petechiae under a maxillary denture are not true purpura. They result from candidal infection and the resulting inflammation of the orifices of accessory salivary glands, not because of denture-created negative pressure as previously believed.

Ecchymosis (common bruise) is an area of extravasated blood usually >1 cm in diameter. They range in color from red-purple (i.e., early) to blue-green-brown (i.e., days later). Careful physical evaluation may reveal the cause to be trauma, hemostatic disorders, Cushing disease, amyloidosis, neoplastic disease, primary idiopathic or secondary thrombocytopenic purpura, or use of anticoagulant drugs such as aspirin, warfarin (Coumadin), or heparin.

Hematomas (large bruise) are large pools of extravasated blood resulting from trauma that results in a palpable mass. They occur commonly in the oral cavity as a result of a blow to the face, tooth eruption, or damage to the posterior superior alveolar vein during administration of local anesthesia (Figs. 56.3 and 56.4). They are usually dark red-brown and tender to palpation. Purpura fade with time and require no specific treatment. Determining the underlying cause is the prime consideration.

Varicosity (Varix) (Fig. 60.5) A **varix** is a dilated vein frequently seen in elderly persons. The swelling is caused by reduced elasticity of the vascular wall as a result of aging or by an internal blockage of the vein. The ventrolateral surface of the anterior two thirds of the tongue is a common location. The floor of mouth, lip, and labial commissure are other common sites. Varices appear dark red to blue-purple. They are usually single, round, dome shaped, and fluctuant. Palpation of the lesion disperses

the blood from the vessel and causes the area to blanch. Thus, the lesions are diascopy-positive.

Varices are benign and asymptomatic and require no treatment. If they are of cosmetic concern, varices can be surgically removed without significant bleeding. Varices are sometimes slightly firm because of fibrotic changes. Thrombosis is a rare complication that produces a firm nodule within the varix. When several veins on the ventral tongue are prominent, the condition is called phlebectasia linguae, or caviar tongue.

Thrombus (Fig. 60.6) A **thrombus** is the formation of a blood clot in a blood vessel. The series of events that includes trauma, activation of the clotting sequence, and formation of a blood clot typically results in the cessation of bleeding. Several days later, plasminogen initiates clot breakdown, and normal blood flow resumes. In certain cases, if the clot does not dissolve, blood flow stagnates and a thrombus is formed.

Intraoral thrombi appear as raised red-brown or blue round nodules, typically in the labial mucosa. They are firm to palpation and may be slightly tender. No sexual predilection is evident, but thrombi are most commonly seen in patients older than 30 years. Thrombi concentrically enlarge to occlude the entire lumen of the vessel or mature and *calcify* to form a **phlebolith**. Phleboliths are rare oral findings that develop in the cheek, lips, or tongue. Radiographs show phleboliths to be doughnut-like, circular, radiopaque foci with a radiolucent center.

Hemangioma (Figs. 60.7 and 60.8) **Hemangiomas** are benign tumors of blood vessels (endothelial cells) that proliferate. They occur early in life, more commonly in women than in men, and develop in any soft tissue or bony intraoral location. They are divided into two forms: **capillary** (consisting of small fine vessels) or **cavernous** (consisting of large thin-walled vascular spaces).

Soft tissue hemangiomas occur commonly in the dorsum of the tongue, gingiva, and buccal mucosa. When situated deep within the connective tissue, they do not alter the color of the mucosal surface. Superficial hemangiomas, in contrast, are red, blue, or purple; flat or slightly elevated; smooth-surfaced; and somewhat firm. Hemangiomas are positive to diascopy and may vary in size from a few millimeters to several centimeters. The borders are usually diffuse. Lobular surfaces are infrequent. Single hemangiomas are most common, whereas multiple lesions are seen in Maffucci syndrome. Facial and oral hemangiomas form a component of **Sturge-Weber syndrome**.

Congenital hemangiomas typically involute. Surgical excision, sclerosing agents, cryotherapy, and radiation therapy have been used to eliminate large and persistent lesions. The differential diagnosis should consider its malignant counterpart (hemangioendothelioma) and Kaposi sarcoma (Fig. 82.7), another malignant vascular tumor associated with immune suppression, aging, and human herpesvirus-8 infection.



Fig. 60.1. Petechiae: caused by viral illness and coughing.



Fig. 60.2. Ecchymosis: in a patient receiving heparin.



Fig. 60.3. Hematoma: after trauma from falling.*

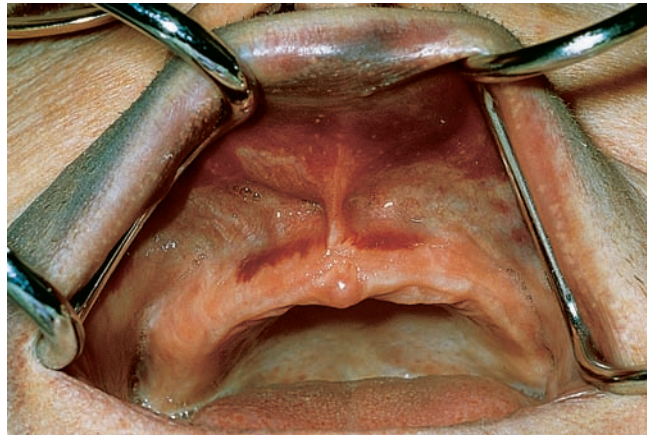


Fig. 60.4. Hematoma: of maxillary mucosa and gingiva.*



Fig. 60.5. Varix: diascopy positive.



Fig. 60.6. Thrombus: of labial mucosa within a varix.



Fig. 60.7. Hemangioma: of ventral tongue.



Fig. 60.8. Hemangioma: of buccal mucosa.

RED LESIONS

Hereditary Hemorrhagic Telangiectasia (Osler-Weber-Rendu Syndrome) (Figs. 61.1–61.4) **Hereditary hemorrhagic telangiectasia** is a genetic disorder that causes abnormalities of blood vessels such that some arteries flow directly into veins rather than into capillaries. This autosomal dominant disease is caused by a defect in a transmembrane protein (endoglin or activin receptorlike kinase-1) that helps form the receptor complex for transforming growth factor beta (TGF- β), which is required for blood vessel wall integrity. These defects result in dilation of terminal blood vessels of the skin and mucous membranes.

Hereditary hemorrhagic telangiectasia is characterized by numerous telangiectasias, which appear as red—purple macules of the skin, mucosa, and other tissues and organs. The lesions are usually 1 to 3 mm, lack central pulsation, and blanch on diascopy. The telangiectasias begin early in life. After puberty, the size and number of lesions tend to increase with age. Men and women are affected equally. Bleeding, such as bleeding from the nose (epistaxis), is a prominent and early feature of this disease.

History, clinical appearance, and histologic features are important in making the diagnosis of hereditary hemorrhagic telangiectasia, as the disease is asymptomatic in many victims. Clinically evident lesions are located immediately subjacent to the mucosa and are easily traumatized, resulting in rupture, hemorrhage, and ulcer formation. Skin lesions are less subject to rupture because of the overlying cornified epithelium. The most common locations on the skin are the palms, fingers, nail beds, face, and neck. Mucosal lesions can be found on the lips, tongue, nasal septum, and conjunctivae. The gingiva and the hard palate are less commonly involved. Vascular malformations also involve the lung, brain, and gastrointestinal tract, especially the liver. Complications are associated with bleeding at various sites, including **epistaxis** from drying or irritation of involved nasal mucosa or from nasotracheal intubation; gastrointestinal bleeding, **melena**, and iron deficiency anemia caused by rupture of telangiectasias of gastrointestinal mucosa and prolonged bleeding; and **hematuria** caused by rupture of telangiectasia within the urinary tract. Other complications include cirrhosis of the liver, pulmonary arteriovenous fistulae leading to pulmonary hypertension, and brain abscesses and emboli. Precautions are recommended with the use of inhalation analgesia, general anesthesia, oral surgical procedures, and hepatotoxic and antihemostatic drugs. Rupture of a telangiectasia may cause hemorrhage that is best controlled by pressure packs. Because brain abscesses form in a small percentage of patients, antibiotic prophylaxis before invasive dental treatment may be beneficial to these patients. Identification of this disorder warrants screening of family members by a physician.

Sturge-Weber Angiomatosis (Sturge-Weber or Encephalotrigeminal Syndrome) (Figs. 61.5–61.8) **Sturge-Weber angiomatosis** is a rare, nonhereditary disorder present at birth characterized by a port-wine birthmark and neurological abnormalities. The syndrome results from the persistence of a vascular plexus and produces four prominent features: (1) venous angiomas of the leptomeninges of the brain, (2) a port-wine stain of the face, (3) neuromuscular deficits, and (4) oculo-oral lesions.

The port-wine stain or nevus flammeus, a macular hemangioma, is the most striking feature of the syndrome. The facial hemangioma is well demarcated, flat or slightly raised, usually unilateral, and red to purple in color. It blanches under pressure. The stain is present at birth, is distributed along one or several branches of the trigeminal nerve, and typically extends to the patient's midline without crossing to the other side. The ophthalmic division of the trigeminal nerve is most frequently affected. No tenderness or inflammation is associated with the hemangioma, and it does not enlarge with age. In many patients with solitary congenital port-wine angiomas of the face, lesions regress spontaneously at puberty. Approximately 20% of persons with facial port-wine stains have Sturge-Weber angiomatosis. The remainder are free of the other features of the syndrome. Involvement of the ophthalmic branch of the trigeminal nerve appears to be most predictive of full involvement of the syndrome.

The altered venous blood flow caused by an angioma of the leptomeninges can result in degeneration of the cerebral cortex, seizures, mental retardation, and hemiplegia. On lateral skull radiographs, gyriiform calcifications characteristically appear as double-contoured “tramlines.” Approximately 30% of patients have ocular abnormalities, including angiomas, colobomas, or glaucoma.

Vascular hyperplasia involving the buccal mucosa and lips is the most frequent oral finding. The palate, gingiva, and floor of the mouth may also be affected. The bright red oral patches are located on areas supplied by the branches of the trigeminal nerve. Like facial lesions, these patches stop abruptly at the midline. Involvement of the gingiva may produce edematous tissue and cause difficulty with hemostasis when surgical procedures involving these tissues are performed. Abnormal tooth eruption, enlarged lips (macrocheilia), large teeth (macrodontia), and enlarged tongue (macroglossia) are sequelae of the vascular overgrowths. Gingival overgrowth may result from phenytoin therapy, which is given because these patients are subject to seizures. Careful assessment of the enlarged gingiva on the ipsilateral side, including biopsy, may be required to establish vascular involvement or drug-induced gingival overgrowth. In areas of vascular hyperplasia, oral surgery should be performed in accordance with strict hemostatic measures.



Fig. 61.1. Hereditary hemorrhagic telangiectasia: lips.*



Fig. 61.2. Hereditary hemorrhagic telangiectasia: gingivae.*



Fig. 61.3. Hereditary hemorrhagic telangiectasia.



Fig. 61.4. Hereditary telangiectasia: diascopy positive.



Fig. 61.5. Sturge-Weber angiomatosis.† port-wine stain.



Fig. 61.6. Sturge-Weber angiomatosis.† oral involvement.



Fig. 61.7. Sturge-Weber angiomatosis: up to palate midline.

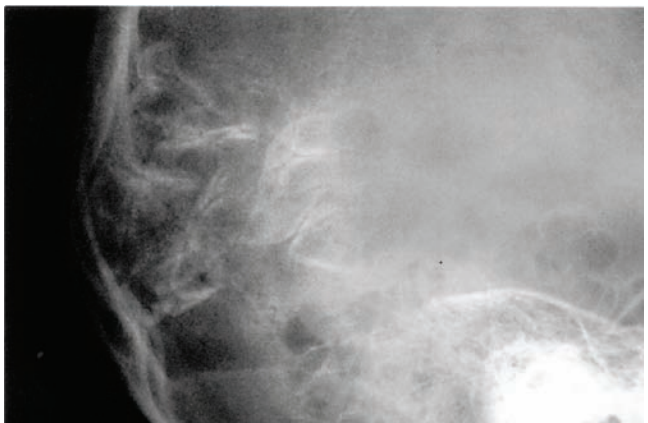


Fig. 61.8. Sturge-Weber angiomatosis: cranial calcifications.

RED AND RED-WHITE LESIONS

Erythroplakia (Figs. 62.1–62.4) **Erythroplakia** is defined as a persistent red patch that cannot be characterized clinically as any other condition. Like leukoplakia, the term, has no histologic connotation. However, unlike leukoplakia, most erythroplakias are histologically diagnosed as epithelial dysplasia or worse and have a much higher propensity for progression to carcinoma. Erythroplakias are most prevalent in the mandibular mucobuccal fold, oropharynx, tongue, and floor of the mouth, and are often associated with tobacco or alcohol use. They are usually asymptomatic. The redness of the lesion is a result of atrophic mucosa overlying a highly vascular (reddish) and inflamed submucosa. The border is often well demarcated. There is no sex predilection, and patients older than 55 years are most commonly affected.

Three clinical variants of erythroplakia are recognized: (1) the **homogeneous** form, which is completely red; (2) **erythroleukoplakia**, which mainly has red patches interspersed with occasional white areas; and (3) **speckled erythroplakia**, which contains white specks or granules scattered throughout the red lesion. Biopsy is mandatory for all types of erythroplakia because 91% represent severe dysplasia, carcinoma in situ, or invasive squamous cell carcinoma. Inspection of the entire oral cavity is required because 10% to 20% of these patients have several erythroplakic areas, a phenomenon known as **field cancerization**.

Erythroleukoplakia and Speckled Erythroplakia (Fig. 62.5) **Erythroleukoplakia** and **speckled erythroplakia**, or speckled leukoplakia, as some prefer, are precancerous red and white lesions. Both are usually asymptomatic. Erythroleukoplakia and speckled erythroplakia have a male predilection, and most are detected in patients older than 50 years. They may occur at any intraoral site but frequently affect the lateral border of the tongue, buccal mucosa, and soft palate. Lesions are often associated with heavy smoking, alcoholism, and poor oral hygiene.

Fungal infections are common in speckled erythroplakias. *Candida albicans*, the predominant organism, has been isolated in most cases. Thus, management of these lesions should include analysis for candida. The cause-and-effect relationship between candidiasis and speckled leukoplakia is unknown, but erythroplakia with leukoplakic regions confers a high risk for atypical cytologic changes and progression to carcinoma.

Squamous Cell Carcinoma (Figs. 62.6–62.8) **Squamous cell carcinoma** is an invasive malignancy of oral epithelium. It is the most common type of oral cancer, accounting for more than 90% of all malignant neoplasms of the oral cavity. Oral cancer may occur at any age, but it is primarily a disease of the elderly. Recent studies demonstrate that 90% of oral cancers occur in persons older than 45 years, and the prevalence is higher in men than women (2.6:1).

The exact cause of oral squamous cell carcinoma is unknown but appears to involve mutations of genes on chromosomes 3 and 9 (*p53*, *ras*) that regulate cell proliferation and death (apoptosis). Cytological changes occur with excessive use of tobacco and alcohol, and may be influenced by microbial infection with human papillomavirus, *Treponema pallidum*, or *C. albicans*. Other contributory factors are those associated with aging, immune compromise, poor nutrition, oral neglect, chronic trauma, and ultraviolet radiation.

In the United States, the most common site of intraoral squamous cell carcinoma is the lateral border and ventral surface of the tongue, followed by the oropharynx, floor of the mouth, gingiva, buccal mucosa, lip, and palate. The buccal mucosa is a common site in persons of developing countries who chronically use quid (betel nut) tobacco. The occurrence of carcinoma of the lip has decreased dramatically in the past decade because of the increased use of protective sunscreen agents. The dorsal surface of the tongue is almost never affected.

The appearance of squamous cell carcinoma is variable; more than 90% of cases are erythroplakic, and about 60% have a leukoplakic component. A combination of colors and surface patterns—such as a red and white lesion that is exophytic, infiltrative, or ulcerated—indicates instability of the oral epithelium and is highly suggestive of carcinoma. Early lesions are often asymptomatic and slow growing. As the lesion develops, the borders become diffuse and ragged, and induration and fixation ensue. If the mucosal surface becomes ulcerated, the most frequent oral symptom is that of a persistent sore or irritation that fails to heal. Advancing disease can cause numbness, swelling, or difficulty in speaking or swallowing. Lesions can extend to several centimeters in diameter if treatment is delayed. This delay permits lesions to invade and destroy vital tissues.

Squamous cell carcinoma spreads by local extension or by way of lymphatic vessels. Affected patients may have palpable regional (submandibular or anterior cervical) lymph nodes. These nodes can be large, firm, rubbery, and possibly fixed to underlying tissue. Staging of the tumor according to the TNM system—**size (T)**, **regional lymph nodes (N)**, and **distant metastases (M)**—allows assessment of the extent of disease. Surgery and radiation therapy have been the principal forms of treatment for oral cancer. The prognosis for oral cancer depends, in large measure, on the site involved (posterior tumors having worse prognosis), the clinical stage at the time of diagnosis and treatment, tumor diameter, the patient's access to adequate health care, and the patient's ability to cope and mount an immunologic response. Because early treatment is paramount, biopsy should be done if neoplasia is suspected. Use of vital stains and new imaging devices can help in early recognition and speed the diagnostic process.

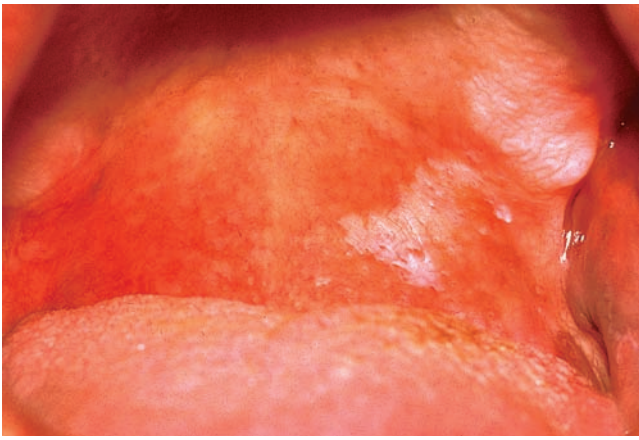


Fig. 62.1. Erythroplakia: seen after tongue was depressed.*

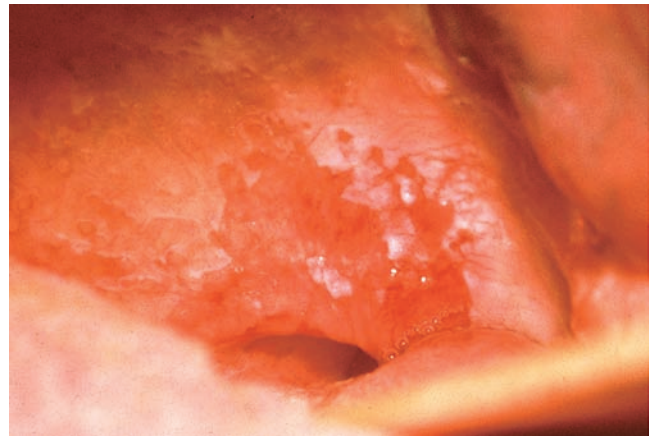


Fig. 62.2. Erythroplakia: on soft palate; tongue depressed.*



Fig. 62.3. Erythroplakia: of floor of mouth.



Fig. 62.4. Carcinoma appearing as erythroplakia.

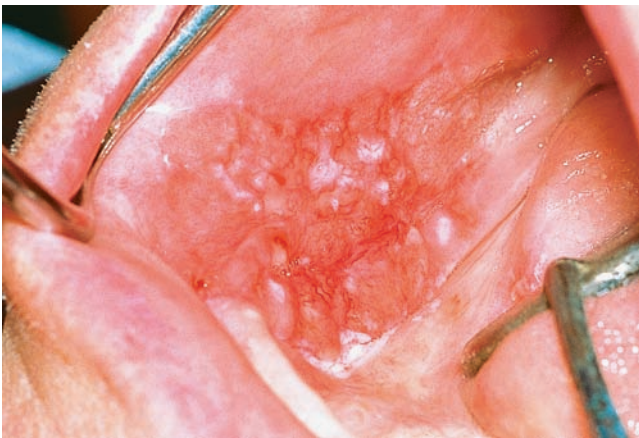


Fig. 62.5. Erythroleukoplakia: squamous cell carcinoma.†



Fig. 62.6. Squamous cell carcinoma: ulcer ventral tongue.†

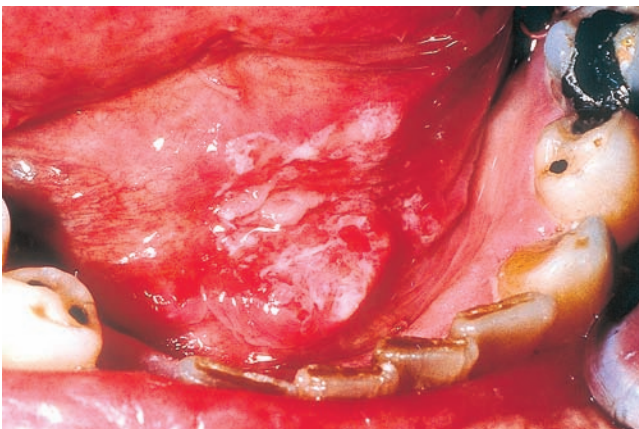


Fig. 62.7. Speckled erythroplakia: squamous cell carcinoma.

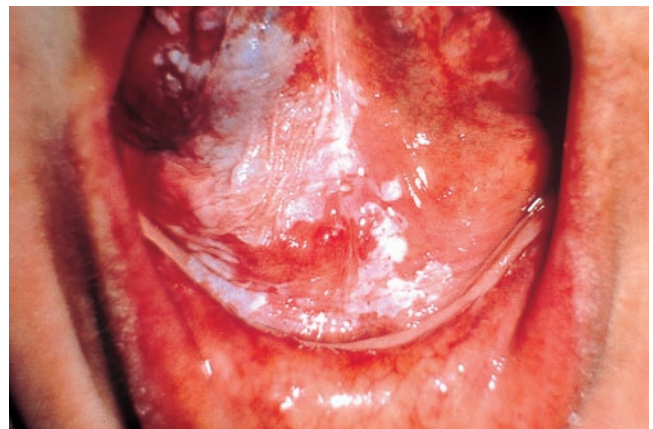


Fig. 62.8. Squamous cell carcinoma: floor of mouth.

RED AND RED-WHITE LESIONS

Lichen Planus (Figs. 63.1–63.6) Lichen planus is a common skin disease that can appear at mucosal sites. The cause and pathogenesis are unknown, but evidence suggests that it is an immunologic disorder in which T lymphocytes are attracted to an antigen within the epithelium. Both CD4 and CD8 T-cell subsets are found heavily dispersed at the epithelial-connective tissue interface of the diseased tissue. This chronic inflammatory state leads to epithelial changes, excessive amounts of fibrinogen deposited at the basement membrane, and eventual destruction of the basal cell layer of the affected epithelium. Nervous, high-strung persons and persons infected with hepatitis C virus are predisposed to lichen planus. Most patients are women older than 40 years. The disease exhibits a protracted course with periods of remission and exacerbation.

The skin lesions of lichen planus are classically described as *purple, polygonal, pruritic papules*. Initially they consist of small, flat-topped, red papules with a depressed central area. The lesions may enlarge and become polygonal in shape or coalesce into larger plaques. The papules progressively acquire a violaceous hue and surface lichenification, which consists of fine white striae. Skin lesions usually itch and may change color to yellow or brown before resolution. Bilateral distribution on the flexor surfaces of the extremities is common, occasionally involving the fingernails, causing dystrophic changes. The vulva or glans penis are sometimes affected.

Oral lesions of lichen planus may have one of four appearances: **atrophic, erosive, striated (reticular), or plaquelike**. More than one form may affect a single patient. The most frequently affected site is the buccal mucosa. The tongue, lips, palate, gingiva, and floor of the mouth may also be affected. Bilateral and relatively symmetric lesions are common. Patients with reticular oral lichen planus characteristically have several delicate white lines and tiny papules arranged in a lacy, weblike network known as **Wickham striae**. The glistening white areas are often asymptomatic but may be of cosmetic concern. They may involve large areas.

Atrophic lichen planus results from atrophy of the epithelium and predominantly appears as a red, nonulcerated mucosal patch. Wickham striae are often present at the border of the lesion. When the attached gingiva is affected, the term **desquamative gingivitis** has been used.

Erosive lichen planus occurs if the surface epithelium is completely lost and erosion results. The buccal mucosa and tongue are commonly affected sites. A vesicle or bulla may initially appear. This eventually breaks down and produces an erosion. Mature lesions have irregular red borders, a yellowish necrotic central pseudomembrane, and an annular white patch, often at the periphery. The condition is intermittently painful and may develop rapidly. All of these features are helpful in differ-

entiating oral lichen planus from other lesions with a similar clinical appearance, such as leukoplakia, erythroplakia, candidiasis, lupus erythematosus, pemphigoid, and erythema multiforme.

The least common type of lichen planus is the asymptomatic **plaque form**. This lesion is a solid white plaque or patch that has a smooth to slightly irregular surface and an asymmetric configuration. Lesions are commonly found on the buccal mucosa or tongue. Patients may be unaware of the lesions.

In many cases, clinical appearance alone can confirm the diagnosis of oral lichen planus, and biopsy is not necessary. Asymptomatic intraoral lesions can be left alone. Biopsy of the atrophic or erosive form should be performed at the border of the lesion, away from areas of ulceration.

Oral lesions of lichen planus tend to be more persistent than those of the skin. A vacation, change in routine, or discharge of psychologically burdensome problems can bring about abrupt and dramatic resolution of the lesions. Chronic, symptomatic, erosive lichen planus lesions are best managed with topical steroids or short courses of systemic steroids and immunosuppressant agents. A few patients with oral lichen planus are diabetic and should be tested for glucose intolerance. Carcinomatous transformation has been reported (in a limited number of cases) to have an association with erosive lichen planus and tobacco use. This relationship, however, remains controversial.

Lichenoid Mucositis (Lichenoid Drug Reaction/Eruption) (Figs. 63.7 and 63.8) **Lichenoid mucositis** closely resembles lichen planus. This disorder is more apparent after 30 years of age and frequently occurs on the buccal mucosa, immediately adjacent to a metallic material (usually an older or corroding restoration). Mild cases are asymptomatic, whereas erosive cases can cause a burning type of pain. The histologic features of this lesion mimic those of lichen planus. Current evidence suggests that the disorder results from a delayed hypersensitivity reaction to antigens in various substances (such as metals, particularly mercury). Interestingly, lichenoid drug reactions can be caused by the systemic administration or application of the same metals (mercury and gold) found in dental restorations. Other drugs shown to induce lichenoid eruptions include angiotensin-converting enzyme inhibitors, antimalarials, beta blockers, nonsteroidal anti-inflammatory drugs, sulfasalazine, and sulfonyleurea compounds (Figs. 64.5–64.8). Also, cinnamon (in chewing gum or food) can induce lichenoid mucositis at the site of contact. Patch testing, to confirm contact allergens, aids in the diagnosis. Treatment consists of removing the causative agent. If a restoration is to be replaced, a different restorative material, preferably porcelain, or composite materials should be used. The prognosis is excellent, and healing occurs within weeks of removing the offending agent.



Fig. 63.1. Lichen planus: violaceous skin plaque of wrist.

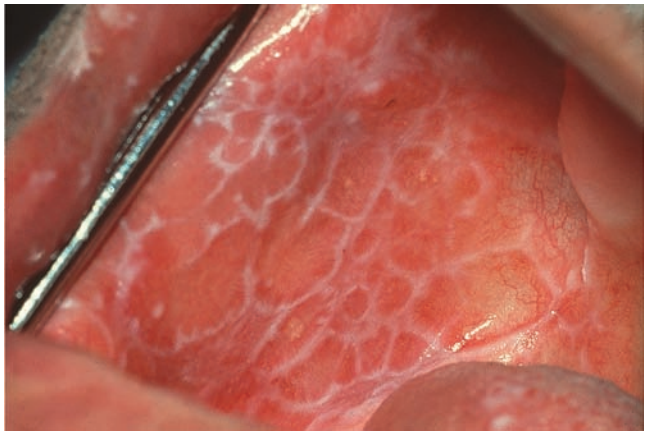


Fig. 63.2. Reticular lichen planus: striae on buccal mucosa.



Fig. 63.3. Erosive lichen planus: of buccal mucosa.*



Fig. 63.4. Erosive lichen planus: opposite buccal mucosa.*

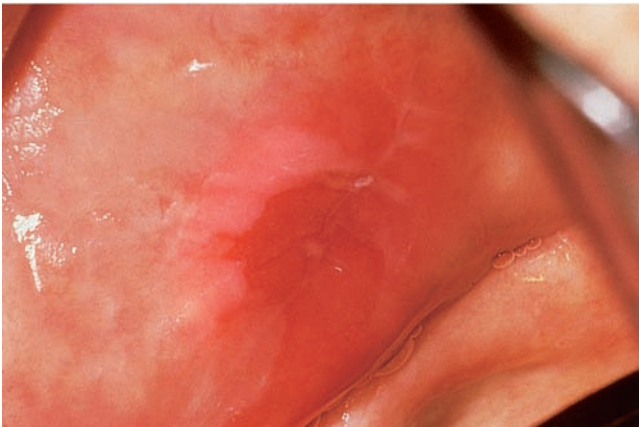


Fig. 63.5. Atrophic lichen planus: of buccal mucosa.



Fig. 63.6. Plaque form of lichen planus.

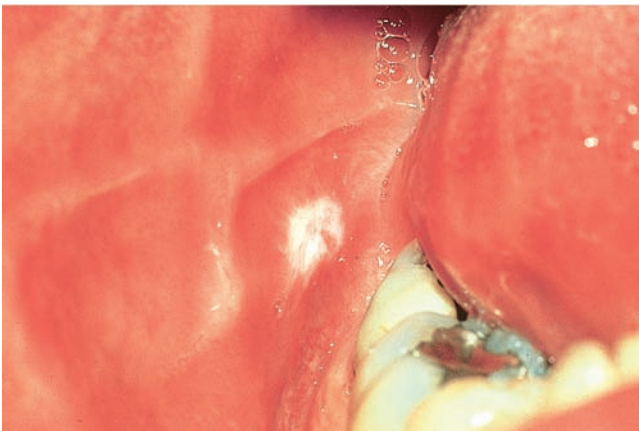


Fig. 63.7. Lichenoid mucositis: adjacent to facial alloy.†

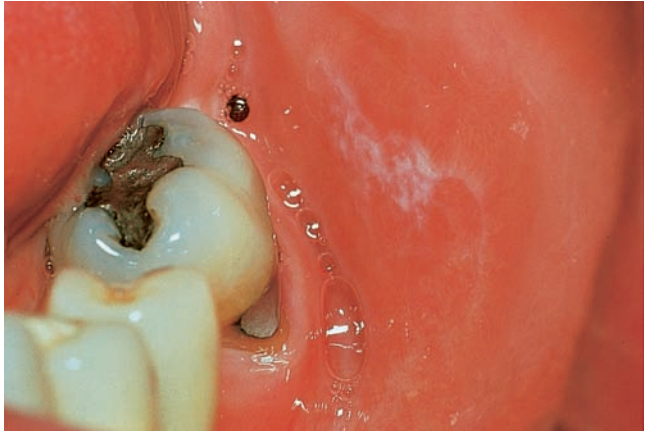


Fig. 63.8. Lichenoid mucositis: opposite buccal mucosa.†

RED AND RED-WHITE LESIONS

Lupus Erythematosus (Figs. 64.1–64.4) **Systemic lupus erythematosus (SLE)** is an autoimmune disorder that develops when the body's immune system attacks the body's own cells and tissue, resulting in inflammation and tissue damage. Affected patients produce antinuclear antibodies (ANA), anti-DNA, and antihistone antibodies against components in the cell nucleus. They also have antibodies directed against the basement membrane of epithelium. All of these antibodies participate in immunologically mediated attack of self-antigens, resulting in tissue injury. Three forms exist: (1) **chronic discoid lupus erythematosus**, also known as **chronic cutaneous lupus erythematosus**, which only involves the skin; (2) **systemic lupus erythematosus**, in which multiple organ systems are involved; and (3) **subacute cutaneous lupus erythematosus**, an intermediate form that produces nonscarring skin lesions, mild musculoskeletal (arthritis) symptoms, and limited or no organ disease. The cause of all three types is unknown.

Chronic cutaneous or **discoid lupus erythematosus**, the benign form of the disease, is a purely mucocutaneous disorder. It may appear at any age but predominates in women older than 40 years. The condition classically produces a red butterfly rash distributed symmetrically on the cheeks across the bridge of the nose. Other prominent sun-exposed areas of the face—including the malar areas, forehead, scalp, and ears—may be involved.

The lesions of lupus erythematosus are chronic, with periods of exacerbation and remission. Mature lesions exhibit three zones: an atrophic center lined by a hyperkeratotic margin, which is surrounded by an erythematous periphery. As the center heals, hypopigmentation (or in some cases hyperpigmentation) results from melanocyte damage at the epidermal-dermal junction. Telangiectasias, blackheads, a fine scale, and hair loss at sites of involvement are common findings. The lesions are usually limited to the upper portion of the body, particularly the head and neck.

Oral lesions are found in 20% to 40% of patients with lupus erythematosus. These lesions may develop before or after skin lesions develop. Lip lesions are red with a white to silvery, scaly margin. A sun-exposed lower lip at the vermilion border is a common site, whereas the upper lip is usually involved as a result of direct extension of dermal lesions. Intraoral lesions are frequently diffuse erythematosus plaques, with erosive, ulcerative, and white components.

Chronic discoid lupus erythematosus sometimes appears as isolated red-white plaques. The buccal mucosa is the most frequent intraoral site, followed by the tongue, palate, and gingiva. The oral lesions are characterized by a central, red atrophic area sometimes covered by a fine stippling of white dots. The peripheral margins are irregular and are composed of alternating red and keratotic white lines extending for a short length up to about 1 cm in a radial pattern. These lesions may mimic lichen planus, but concurrent ear involvement helps to exclude the diagnosis of lichen planus. Ulcerative lesions

are painful and require treatment. Avoidance of emotional stress, cold, sunlight, and hot spicy foods is necessary. The use of sunscreens, topical steroids, systemic steroids, and antimalarial and immunosuppressive agents have proven effective. Patients using antimalarial agents require close ophthalmologic follow-up.

Systemic manifestations of the disease often begin with reports of fatigue, fever, and joint pain. Generalized nontender lymphadenopathy is often present. Hepatomegaly, splenomegaly, peripheral neuropathy, and hematologic abnormalities may be seen. Strict avoidance of sun exposure is necessary because sunburn can trigger acute exacerbations. Involvement of the kidneys and heart is a common occurrence that may prove fatal. Skin and oral lesions may accompany the condition, but there is little chance of conversion from discoid to systemic lupus. Patients with systemic lupus erythematosus often have other concurrent autoimmune collagen-vascular diseases, such as Sjögren syndrome and rheumatoid arthritis. Allergic mucositis, candidiasis, leukoplakia, erythroleukoplakia, and lichen planus must be considered in the differential diagnosis of oral lupus erythematosus lesions. Biopsy and histologic examination with immunofluorescence confirm the diagnosis. Precautions are advised in the dental treatment of patients with lupus erythematosus, who may be taking high doses of systemic steroids, because of their predisposition to delayed wound healing, risk for infection, and the possibility of stress-induced adrenal crisis characterized by cardiovascular collapse. In addition, these patients are at risk for cardiomyopathy and defective heart valves.

Lichenoid and Lupuslike Drug Eruption (Figs. 64.5–64.8) **Stomatitis medicamentosa** is a general term used to describe an oral hypersensitivity reaction to a drug that results in oral lesions. Two subcategories of oral hypersensitivity, called **lichenoid drug eruption** and **lupuslike drug eruption**, have been described. They produce reticular or erosive lesions similar in appearance to lichen planus and lupus erythematosus. Although the appearance may vary, white linear plaques with red margins are common. The lesions may erupt immediately or after prolonged use of a drug. Persistent inflammatory changes may result in large erythematous areas, mucosal ulceration, and pain. Drug-induced lupus erythematosus is often associated with arthritis, fever, and renal disease. Hydralazine and procainamide are the most common instigators of lupuslike drug eruptions. Other drugs known to cause lupuslike eruptions include gold, griseofulvin, isoniazid, methyldopa, penicillin, phenytoin, procainamide, streptomycin, and trimethadione. Drugs known to induce lichenoid eruptions include chloroquine, dapsone, furosemide, gold, mercury, methyldopa, palladium, penicillamine, phenothiazines, quinidine, thiazides, certain antibiotics, and heavy metals. Consultation with a physician and withdrawal of the offending medication leads to regression of the lesion(s). A substitute drug is usually selected to manage the patient's systemic problem.



Fig. 64.1. Discoid lupus erythematosus: butterfly rash.



Fig. 64.2. Discoid lupus erythematosus: sun-exposed area.



Fig. 64.3. Alternating red-white lines of lupus erythematosus.

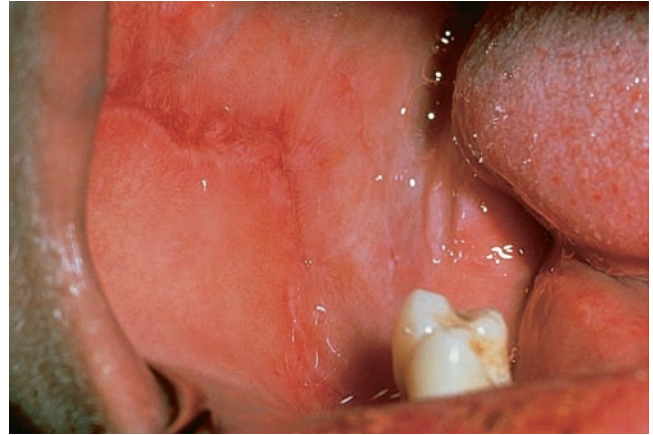


Fig. 64.4. Atrophic lesion of lupus erythematosus.



Fig. 64.5. Lupuslike drug eruption: with amitriptyline use.*



Fig. 64.6. Lupuslike drug eruption: opposite buccal mucosa.*



Fig. 64.7. Lichenoid drug eruption: lateral tongue.†



Fig. 64.8. Lichenoid drug eruption: after drug withdrawal.‡

RED AND RED-WHITE LESIONS

Pseudomembranous Candidiasis (Thrush) (Figs. 65.1 and 65.2)

Pseudomembranous candidiasis is an opportunistic infection caused by overgrowth of the superficial fungus *C. albicans*. It appears as diffuse, velvety, white mucosal plaques that are nonpainful until they are wiped off, leaving a red, raw, or bleeding surface. The organism is a common inhabitant of the oral cavity, gastrointestinal tract, and vagina. Infants whose mothers have vaginal thrush at the time of birth and adults who have experienced a change in normal oral microflora because of antibiotics, steroids, or systemic alterations such as diabetes, immunodeficiency, or chemotherapy are frequently affected. There is no racial or sex predilection. Pseudomembranous candidiasis is usually found on the buccal mucosa, tongue, and soft palate. In patients with asthma who use a steroid inhaler, the pattern appears as a circular or oval reddish white patch at the site of aerosol contact on the palate. Diagnosis is made by clinical examination, fungal culture, or direct microscopic examination of tissue scrapings. A cytologic smear treated with potassium hydroxide, Gram, or periodic acid–Schiff (PAS) stain will reveal budding organisms with branching pseudohyphae. Topical or systemic antifungal medication for 2 weeks usually produces resolution.

Chronic Hyperplastic Candidiasis (Figs. 65.3 and 65.4) **Chronic hyperplastic candidiasis** is caused by candidal organisms that penetrate the mucosal surface and stimulate a hyperplastic response. Chronic irritation, poor oral hygiene, and xerostomia are predisposing factors. Thus, smokers and denture wearers are commonly affected. Diabetes mellitus and HIV infection can also be contributory. Sites most affected are the dorsum of the tongue, palate, buccal mucosa, and labial commissures. The lesion invariably has a distinctive raised border, a white or grayish pebbly surface, and red zones caused by destruction of the mucosa. Thus, the condition may resemble leukoplakia, erythroleukoplakia, or verrucoid growths.

Chronic hyperplastic candidiasis cannot be peeled off. Thus, the diagnosis must be made by biopsy. On microscopy, the organisms may be identified by routine hematoxylin and eosin stain or, more appropriately, by PAS stain. With adequate topical application of an antifungal agent, the condition usually resolves. In some instances, surgical stripping may be required. All patients with hyperplastic candidiasis should be followed closely because this form may be related to speckled erythroplakia, a lesion that is often premalignant or worse.

Erythematous Candidiasis. There are several “red”-appearing forms of candidiasis. Three are discussed here; median rhomboid glossitis (a fourth form) is discussed elsewhere (Figs. 47.2–47.3).

Acute Atrophic Candidiasis (Antibiotic Sore Mouth) (Fig. 65.5)

The use of broad-spectrum antibiotics, particularly tetracyclines, or topical steroids can result in acute atrophic

candidiasis. This fungal infection is the result of an imbalance in the oral ecosystem between *Lactobacillus acidophilus* and *C. albicans*. Antibiotics taken by the patient can reduce the *Lactobacillus* population and permit candidal organisms to flourish. The infection produces desquamated areas of surface mucosa that appear as diffuse, atrophic red patches that cause burning pain. The location of the patches can indicate the cause. Lesions affecting the buccal mucosa, lips, and oropharynx often suggest the systemic use of antibiotics, whereas redness of the tongue and palate are more common after use of antibiotic troches. When the tongue is affected, a surface devoid of filiform papillae is common. Candidiasis rarely affects the attached gingiva. If this is the clinical finding, severe immunosuppression is a distinct possibility. The diagnosis is confirmed by demonstration of budding organisms or hyphal forms on a stained cytologic smear. Treatment should be the withdrawal of offending antibiotics and use of antifungal drugs.

Angular Cheilitis (Fig. 65.6) Angular cheilitis, or *perlèche*—meaning “to lick” is a chronic painful erosive condition involving the labial commissures caused by *C. albicans*, *Staphylococcus aureus*, lip-licking, and pooling saliva. It appears at the corners of the lips as red erosions with central fissures that may ulcer. Erythema, crusting, and brownish granulomatous nodules may occur along the peripheral margins. Discomfort caused by opening the mouth may limit normal oral function. Predisposing factors include poor nutrition, loss of vertical dimension, and high sucrose intake. Treatment involves antifungal agents, correction of predisposing factors, and habit cessation.

Chronic Atrophic Candidiasis (Denture Stomatitis) (Figs. 65.7 and 65.8) **Chronic atrophic candidiasis** or **denture stomatitis** is the most common form of chronic candidiasis. It presents as an asymptomatic red lesion on the palate of complete and partial denture wearers, particularly elderly women who wear their dentures at night. Only rarely are dentate patients and the mandible affected. Misnomers for this disease are denture sore mouth and denture base allergy.

Chronic atrophic candidiasis is caused by candidal organisms under the denture base. There are three stages of denture stomatitis. The earliest lesions are red pinpoint areas of hyperemia limited to the orifices of the palatal minor salivary glands. The second stage produces a diffuse erythema that is sometimes accompanied by epithelial desquamation. **Papillary hyperplasia**, consisting of multiple fibromalike papules, is the third stage. With time, the papules may enlarge to form red nodules. Effective therapy requires antifungal treatment of the mucosa and denture base. Traumatic influences, such as the rocking action of an ill-fitting denture, should be eliminated to speed healing. Occasionally surgical stripping is required.



Fig. 65.1. Acute pseudomembranous candidiasis: in a patient with diabetes.

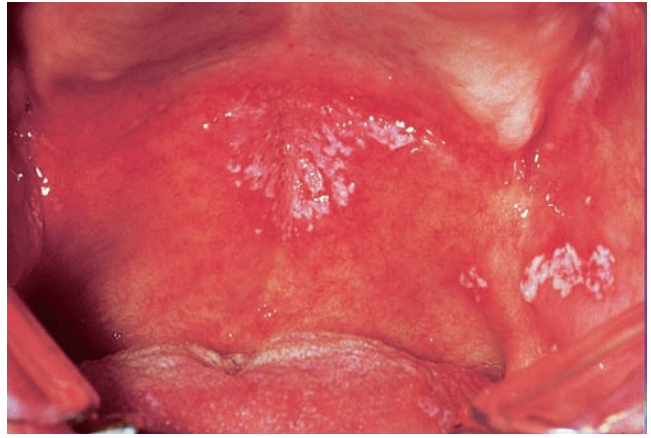


Fig. 65.2. Acute pseudomembranous candida: steroid user.



Fig. 65.3. Chronic hyperplastic candidiasis: at commissures.



Fig. 65.4. Chronic hyperplastic candidiasis: of labial mucosa.



Fig. 65.5. Acute atrophic candidiasis: inhaled steroid use.



Fig. 65.6. Angular cheilitis: after antibiotic therapy.

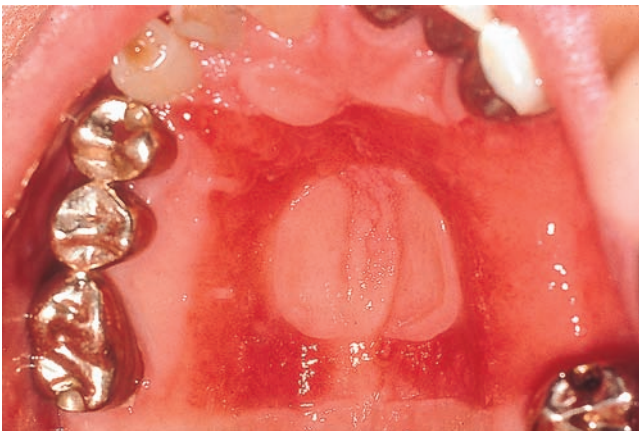


Fig. 65.7. Chronic atrophic candidiasis: denture-bearing area.

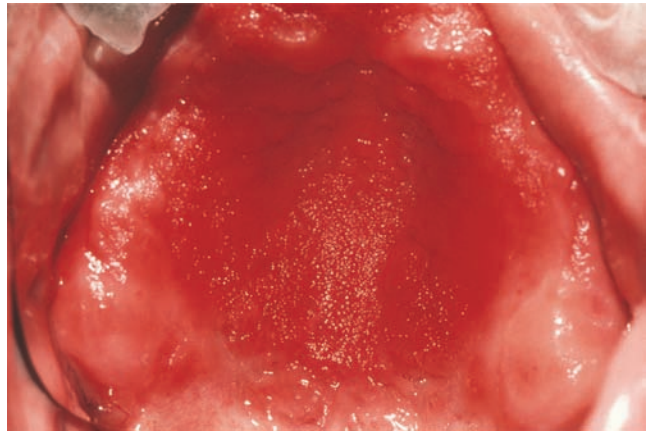


Fig. 65.8. Papillary hyperplasia: third stage.

PIGMENTED LESIONS

Melanoplakia (Physiologic Pigmentation) (Fig. 66.1) **Melanoplakia** is a generalized and constant dark pigmentation of the oral mucosa, commonly seen in dark-skinned persons. The condition is physiologic, not pathologic. It results from increased amounts of melanin (an endogenous pigment) that are deposited in the basal layer of the mucosa and lamina propria. The most common site for observing melanoplakia is the attached gingiva. It often appears as a diffuse, ribbonlike, dark band with a well-demarcated and curvilinear border that separates it from the alveolar mucosa. The region is characteristically symmetric and asymptomatic. Infrequently, it can be patchy or asymmetric. The degree of pigmentation varies from light brown (Fig. 7.10) to dark brown and infrequently may appear blue-black. Other sites of occurrence are the buccal mucosa, hard palate, lips, and tongue. At these sites, the deposition of pigment is often multifocal and diffuse. Melanoplakia requires no treatment, but should be differentiated from similar-appearing conditions that produce oral pigmentations, such as Addison disease, Albright syndrome, Peutz-Jeghers syndrome, heavy metal pigmentation, and antimalarial drugs and other conditions discussed in this section.

Tattoo (Figs. 66.2–66.5) **Tattoos** are caused by intentional or accidental implantation of exogenous pigments into the mucosa. The most common intraoral type is the **amalgam tattoo**. The amalgam tattoo appears as a slate-gray to blue-black, nonelevated discoloration that is usually irregular in shape and variable in size. It results from the entrapment of amalgam in a soft tissue wound, such as an extraction socket or a gingival abrasion from a rotating bur. Deterioration of the metallic compounds of the amalgam impart the characteristic blue-gray color. Focal discolorations may occasionally appear green to dark gray because of the deposition of high copper alloys.

Amalgam tattoos are usually seen in the gingiva in posterior areas adjacent to a large amalgam restoration or gold casting. These lesions are not limited to the gingiva and may also be seen on the edentulous ridge, vestibular mucosa, palate, buccal mucosa, and floor of the mouth. The clinical diagnosis of an amalgam tattoo can be confirmed by finding radiographic evidence of the foreign metal in the paradental tissue (Fig. 66.4). The radiographic appearance may vary from the absence of demonstrable particles to pinpoint or globular radiopacities several millimeters in diameter. If radiographs fail to demonstrate suspected metallic particles or there are no restorations in the vicinity, a biopsy is required to rule out more serious pigmented lesions.

Other types of tattoos seen in the oral cavity are the graphite pencil wound (graphite implantation) and india ink tattoos. Graphite implantation appears as a focal, slate-gray macule after trauma embeds the pencil tip into the mucosa. Sites frequently affected are the lip

and the palate. The nature of the lesion can be easily ascertained by questioning the patient. Ordinary India ink tattoos are occasional findings on the labial mucosa of the lower lip, sometimes conveying a message. In general, tattoos are harmless and of no clinical significance. However, they may be indistinguishable from more potentially ominous lesions.

Ephelis (Freckle) (Fig. 66.6) An **ephelis** is a small, light- to dark-brown macule that appears on the lip or skin after active deposition of melanin triggered by exposure to sunlight. Unlike some pigmentations, this lesion remains nonelevated, essentially unchanged in size (<3 mm) with time, darkens in response to sunlight, and has a predilection for light-skinned or red-headed persons. A single freckle is clinically distinguished from an oral melanotic macule by the history of a traumatic or inflammatory episode that precedes the development of the latter condition. On microscopic examination, an ephelis shows an increase in melanin pigment without an increase in the number of melanocytes. Multiple freckles on the lip should be distinguished from the identical lip ephelides seen in conjunction with palmar pigmentations and intestinal polyposis of **Peutz-Jeghers syndrome** (Figs. 68.1 and 68.2). Ephelides may be of cosmetic concern, but most only require clinical observation.

Smoker's Melanosis (Tobacco-Associated Pigmentation) (Figs. 66.7 and 66.8) Smoking tobacco imparts **smoker's melanosis**, a characteristic change in color to exposed mucosal surfaces. The condition is not a normal physiologic process but instead results primarily from the deposition of melanin in the basal cell layer of the mucosa. The relationship of smoker's melanosis and inflammatory changes that result from heat, smoke inhalation, and the absorption of exogenous pigments has not been fully determined. However, it has been suggested that the deposition of melanocytes is a protective response against the toxic substances in tobacco smoke.

Smoker's melanosis affects older persons who are heavy smokers. It appears as a diffuse smoky-gray-brown patch up to several centimeters in size. Dark-brown foci or zones are often distributed asymmetrically throughout the patch. The mandibular anterior gingiva and buccal mucosa are the most frequently affected sites. Other susceptible sites include the labial mucosa, palate, tongue, floor of the mouth, and lips. The degree of pigmentation ranges from light to dark brown and appears to be directly related to the amount of tobacco smoked. Brown-stained teeth and halitosis usually accompany the condition. Smoker's melanosis itself is not premalignant. However, the clinician should closely inspect the adjacent tissues for other tobacco-induced lesions, which may be more significant. Smoking cessation programs should be offered to these patients.



Fig. 66.1. Melanoplakia: along the attached gingiva.



Fig. 66.2. India ink tattoo: conveying a message.



Fig. 66.3. Amalgam tattoo: in attached gingiva.*

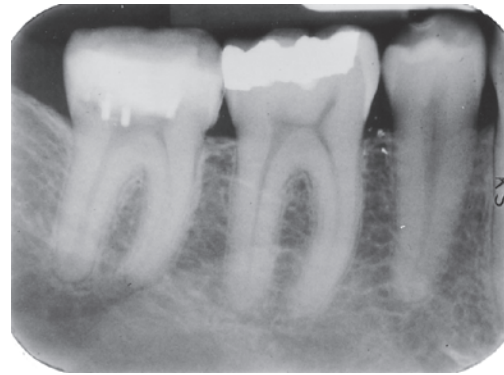


Fig. 66.4. Radiographic evidence of amalgam tattoo.*

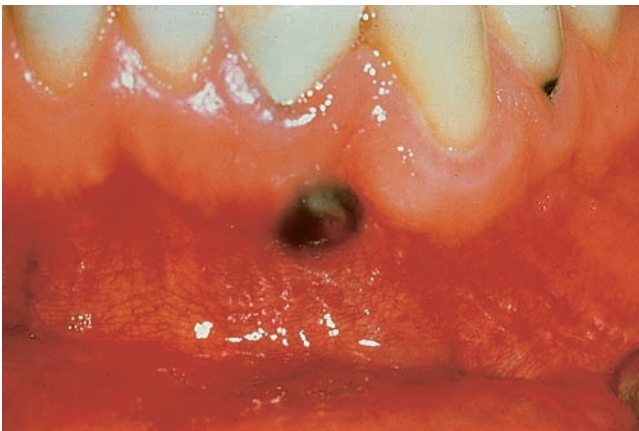


Fig. 66.5. Focal argyrosis: after silver-point endodontics.



Fig. 66.6. Ephelides: multiple freckles of the face and lips.



Fig. 66.7. Smoker's melanosis: of lateral soft palate.



Fig. 66.8. Smoker's melanosis: of the buccal mucosa.

PIGMENTED LESIONS

Oral Melanotic Macule (Focal Melanosis) (Figs. 67.1 and 67.2) An **oral melanotic macule** is a small, flat circumscribed pigmentation of the lip or mouth. It results from focal deposition of melanin along the basal layer of epithelium and superficial layer of connective tissue. These asymptomatic pigmentations are usually single, <1 cm, and common in light-skinned persons between 25 and 45 years of age. They represent reactions to trauma, inflammation, or sun damage. The most common site is the lower lip, close to the midline. Other sites include the gingiva, buccal mucosa, and palate. The color is uniform and may be blue, gray, brown, or black. Biopsy is recommended unless no visible change has occurred in many years. Periodic observation should be provided.

Nevus (Figs. 67.3–67.6) A **nevus** is a flat or sometimes raised growth comprised of a collection of nevus cells (called *theques*) in the epithelium or dermis. It is commonly dark and seen on the skin. Occasionally, it occurs in the oral mucosa. There are many types of nevi, which are broadly classified as either congenital or acquired. Congenital nevi are present at birth and are also known as birthmarks or garment trunk nevi. They are usually larger than acquired nevi and have a higher incidence of malignant transformation.

Acquired nevi, or **moles**, occur later in life and usually appear as dark, slightly raised, papules. They are often symmetrical, uniform in color, but can be pink (amelanotic), brown, grayish, or black. In the mouth, nevi are rare. Most are asymptomatic, small, well-circumscribed, dome-shaped pigmented papules or macules that occur on the palate and buccal mucosa mainly of women. Their size tends to remain constant after puberty.

Benign nevi are classified into four types according to histologic appearance and location of the theques, which extend deeper as the lesion matures. Least to most common are the junctional nevus, compound nevus, blue nevus, and intramucosal nevus.

The **junctional nevus** is an early stage nevus in which the nevus cells are located at the junctional layer of the epithelium and lamina propria. These lesions are the rarest of oral nevi and usually appear flat and brown and have a diameter of <1 cm. The palate and buccal mucosa are common locations.

The **compound nevus**, as the name implies, is composed of nevus cells located in the epithelium and the lamina propria. The compound nevus rarely undergoes malignant transformation.

The **intramucosal nevus** has ovoid nevus cells located in the connective tissue only. This entity is analogous to the intradermal nevus, which appears as a dark, raised papule on the skin, often seen with a hair growing from it. It is rare, however, to find a hair associated with the intramucosal nevus of the mouth. The intramucosal nevus is usually brown, raised, and <0.8 cm in diameter.

The name **blue nevus** originates from the typical blue or blue-black color imparted by the spindle-shaped nevus cells located deep in the connective tissue. It appears as a persistent small, focal blue macule, most commonly on the palate of young adults. Malignant transformation of intraoral blue nevi is very rare.

On rare occasions **Hutchinson freckle** (lentigo maligna) is found in the mouth. The melanotic freckle is usually seen on sun-exposed skin (face) of persons older than 50 years. This lesion is flat, irregularly shaped, with zones of varying brown color. It tends to spread superficially (laterally) and undergo malignant transformation.

Nevi may be difficult to distinguish from their malignant counterparts. Thus, all intraoral pigmented lesions should be biopsied.

Melanoma (Figs. 67.7 and 67.8) A **melanoma** is a malignant tumor that begins in the cells (melanocytes) that produce skin-coloring pigment. Melanomas occur primarily in sun-exposed skin surfaces and infrequently in the oral cavity. They occur about twice as frequently in men as women and mostly in light-skinned persons between 20 and 50 years of age. Melanomas often appear after age 50. There is no sex predilection. About 30% of melanomas arise from previously existing pigmented lesions, such as moles, particularly ones with a history of trauma. They may appear flat or raised, nonpigmented or pigmented. Pigmented lesions are usually deep brown, gray, blue, or jet black. Eighty percent of oral melanomas occur on the palate or maxillary alveolar ridge. Less commonly, the anterior gingivae and labial mucosa are affected. Malignant changes are the result of DNA damage of genes critical for cell cycle control—often induced by ultraviolet light.

Melanoma begins as a small superficial or slightly raised patch that grows slowly and laterally over several months. Early recognizable signs are **A—asymmetrical lesion; B—border irregularity; C—color variation within the lesion; and D—diameter enlarging**. Eventually, a prominent, nonmovable, dark lesion develops. The clinician should be alert to features that include multiple colors (the combination of red together with blue-black is particularly ominous); change in size; satellite lesions arising at the periphery of the lesion; and signs of inflammation, such as a peripheral zone of erythema. Late signs include bleeding and ulceration, firmness, and firm regional lymph nodes. Intraoral melanomas are extremely dangerous and more serious than their cutaneous counterpart because of early and wide metastasis, which results in poor prognosis. Early diagnosis when tumors are <1.5 mm in size and complete resection are critical to long-term survival. The 5-year survival rate of oral melanoma is only about 40%. About 5% of melanomas recur, dictating long-term follow-up care.



Fig. 67.1. Oral melanotic macule: in the lower lip.

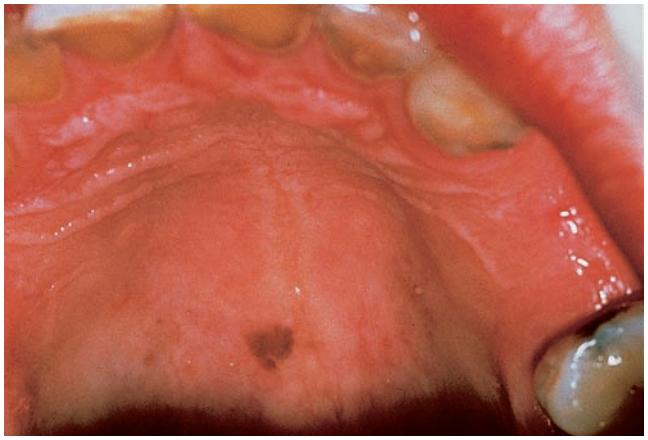


Fig. 67.2. Oral melanotic macule: of the hard palate.

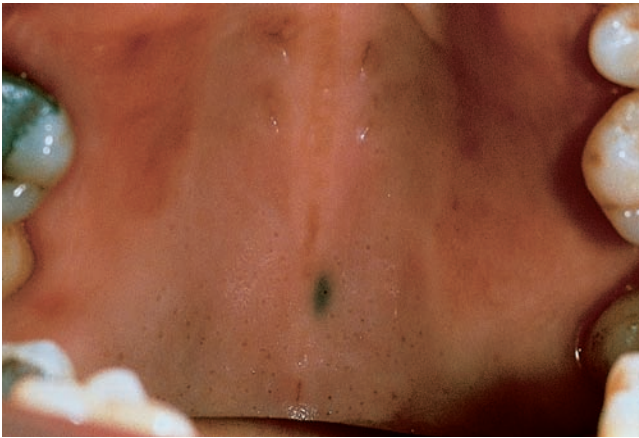


Fig. 67.3. Compound nevus: uniform color, on palate.

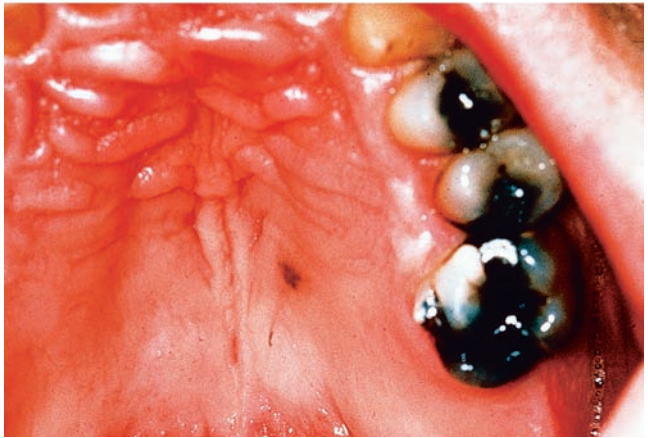


Fig. 67.4. Intramucosal nevus: amelanotic, next to molar.

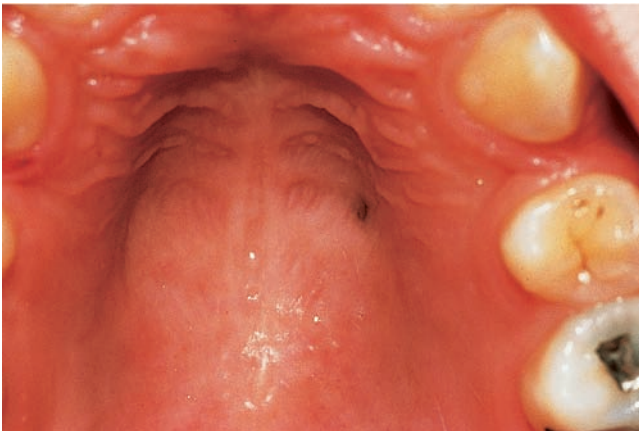


Fig. 67.5. Blue nevus: uniform slate-blue color, hard palate.



Fig. 67.6. Blue nevus: of the lateral palatal vault.

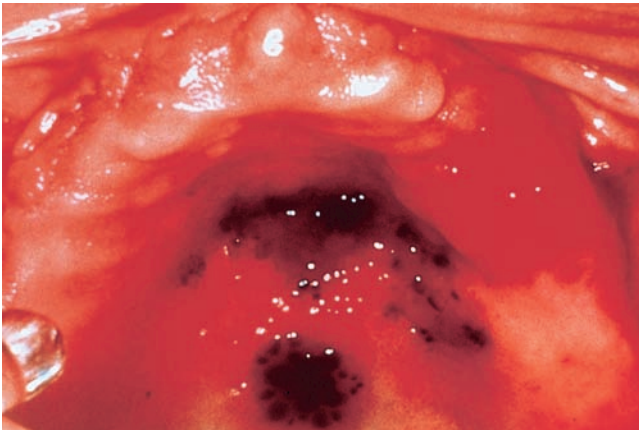


Fig. 67.7. Melanoma: satellite lesions of palate.



Fig. 67.8. Melanoma: color variation, in soft palate and tuberosity.

PIGMENTED LESIONS

Peutz-Jeghers Syndrome (Hereditary Intestinal Polyposis) (Figs. 68.1 and 68.2) **Peutz-Jeghers syndrome** is a genetic condition characterized by multiple pigmented macules and benign polyps, primarily in the intestines. It is an autosomal dominant condition caused by a germ-line mutation in the *LKB1* gene of chromosome 19 that encodes a multifunctional serine-threonine kinase. The macules are distributed on the skin around the eyes, nose, mouth, lips, perineum, oral mucosa, gingiva, and palmar and plantar surfaces of the hands and feet. The multiple benign intestinal polyps are hamartomatous tissue growths that usually occur in the ileum but also are found in the stomach and colon. Symptoms such as intermittent colicky pain and reports of obstruction may be concurrent. The perioral pigmentations must be distinguished from multiple ephelides and lentigines of the LEOPARD syndrome.

The most common intraoral locations for the macules are the lips and buccal mucosa. Characteristically, the density of macules is higher on the vermilion than the adjacent skin. The macules are asymptomatic, small, flat brown ovals that do not darken with exposure to the sun, as freckles do. In contrast to their cutaneous counterparts, the intraoral spots tend to persist into adulthood, whereas the dermal macules may fade with age. No treatment is necessary for the macules, which on microscopic examination contain elongated melanocytes along the basal cell layer and lamina propria. Although the macules are benign, they are of considerable clinical significance because a small percentage of affected patients are prone to develop gastrointestinal (colorectal) adenocarcinoma and are at an increased risk for tumors of the reproductive system. The diagnosis of Peutz-Jeghers syndrome therefore necessitates prompt medical evaluation.

Addison Disease (Adrenal Cortical Insufficiency) (Figs. 68.3 and 68.4) **Addison disease** is an endocrine disorder in which the adrenal gland produces insufficient amounts of steroid hormones. It commonly results from autoimmune-induced destruction of the adrenal gland. Other causes include pituitary insufficiency, tumor invasion, adrenalectomy, infectious diseases, and gram-negative sepsis. Progression of the disease results in anemia, anorexia, diarrhea, hypotension, nausea, salt craving, weakness, and weight loss.

As serum cortisol levels drop, a feedback loop from the adrenal gland to the pituitary gland triggers the production of melanocyte-stimulating hormone by the pituitary gland. This leads to the deposition of melanin in the skin, especially in sun-exposed areas. Classically, the skin acquires a bronze tan that persists after sun exposure. Darkening may be initially noted on the knuckles, elbows, palmar creases, and intraoral mucosa.

In the mouth, the disease is characterized by hypermelanosis that is similar in appearance to physiologic pigmentation. The pattern is not unique and may consist of multiple focal brown-bronze to blue-black spots or gen-

eralized, diffuse streaks of dark-brown pigmentation. The pigmented areas are usually macular, nonraised, brown, and varied in shape. The buccal mucosa and gingiva are most commonly affected, but pigmentation may also extend onto the tongue and lips. Biopsy is not diagnostic, and serum cortisol tests are recommended. Replacement therapy with corticosteroids produces a gradual diminution of the hyperpigmentation. Thus, the degree of oral pigmentation is a sensitive indicator of therapeutic effectiveness. Transient changes in pigmentation in a treated patient may indicate inadequate therapy.

Heavy Metal Pigmentation (Figs. 68.5–68.8) Excessive ingestion of heavy metals (bismuth, lead, mercury, silver) and certain drugs (cis-platinum, antimalarial agents, antipsychotic agents, birth control pills) can produce mucocutaneous pigmentations. Bismuth is commonly found in diarrhea medications, which if used for a long term results in diffuse deposition of the metal in the gingiva. The discoloration is confined to the marginal gingiva, particularly in areas where inflammation is present. The bismuth line usually appears blue to black in a linear distribution along the gingival sulcus. A metallic taste and burning mucosa are common symptoms.

Lead poisoning, or **plumbism**, is usually a result of occupational exposure to excessive doses of lead used in paints, batteries, or plumbing. The most prominent intraoral change and early diagnostic sign is a gray-black lead line that occurs from the deposition of lead sulfide in the marginal gingiva. Spotty gray macules on the buccal mucosa, a coated tongue, neurologic deficits (tremor of the extended tongue), and hypersalivation are other intraoral findings. The condition is reversible if exposure to lead is eliminated.

Mercury poisoning, or **acrodynia**, can be acquired by absorption, inhalation, or ingestion. Although uncommon today, acrodynia was the result of treatment for syphilis earlier in the century. Inadequate mercury hygiene measures, such as handling mercury, breathing mercury vapors, and spilling mercury, place dental personnel at risk for acrodynia. Like bismuth and lead intoxication, mercury poisoning produces a dark mercury gingival line. In addition, the disease is often accompanied by many signs and symptoms, including abdominal pain, anorexia, headaches, insomnia, psychological symptoms, vertigo, oral ulcerations, hemorrhage, a metallic taste, sialorrhea, burning mouth, and periodontal destruction.

Silver pigmentation, or **argyria**, is a rare occurrence that most often results from prolonged exposure to silver-containing ocular, nasal, or oral medications. Asymptomatic pigmentations accumulate in the sun-exposed areas of the skin, along with the hair, fingernails, and oral mucosa. A blue-gray pigmentation is characteristic. Once evident, the pigmentation is irreversible. Nasal inhalation of solutions containing silver salts have a propensity to deposit in the palatal mucosa, imparting a color similar to that seen on the skin. Treatment is immediate withdrawal of the medication.



Fig. 68.1. Peutz-Jeghers syndrome: lips and perioral skin.



Fig. 68.2. Peutz-Jeghers syndrome: of the buccal mucosa.



Fig. 68.3. Addison disease: pigmentation of the lips.*

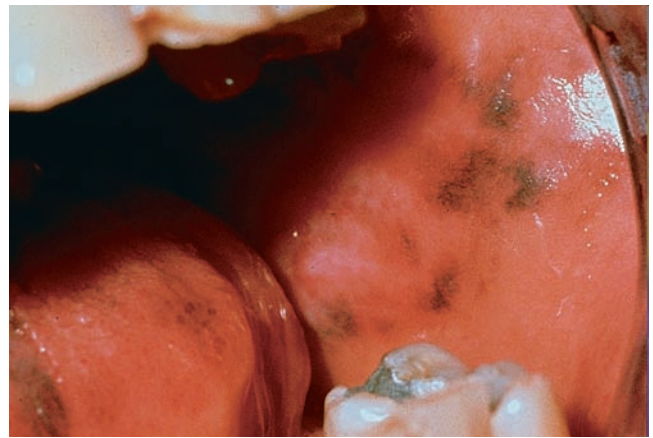


Fig. 68.4. Addison disease: of the buccal mucosa.*



Fig. 68.5. Lead line: along marginal gingiva.



Fig. 68.6. Pigmentation: from silver acetaldehyde detonation.



Fig. 68.7. Argyria: chronic use of silver-containing nose drops.†



Fig. 68.8. Heavy silver pigmentation (argyria): of palate.†

CASE STUDIES

CASE 17. (Fig. 68.9) This 28-year-old Hispanic woman presents to your office for a recall visit. She is good about oral self-care and has nice restorations with no visible decay, but hasn't been in for a while. She claims to smoke four cigarettes per day and has done this for about 11 years. She is not married and dates around. You saw her about 2½ years ago, and the lesion was much smaller then.

1. What is the pink, raised, midline, linear soft tissue structure?
2. Describe the clinical findings (appearance and location).
3. Which term best describes the lesion?
 - A. Leukoplakia
 - B. Erythroplakia
 - C. Speckled erythroplakia
 - D. Morsicatio buccarum
4. What term more specifically describes this lesion?
5. Is this lesion a normal finding, a variant of normal, or disease? What clinical features are suggestive that this condition is benign or malignant?
6. Is this condition associated with smoking? How can you tell if she smokes?
7. If this lesion is removed, do you expect it to resolve or recur?
8. Do you expect this condition to be symptomatic?



CASE 18. (Fig. 68.10A & 68.10B) You take this photograph of a 50-year-old man in your dental office. He has many restorations that were placed at another dental office, and is asymptomatic. He is hypertensive and takes medication for this condition. He recently had a prostate biopsy. The results are pending.

1. Based on the clinical photograph only, with what materials are the molars restored?
2. What part of the tooth is the yellowish component seen at the gingival margin of lower premolar in the clinical photograph? Why is this apparent?
3. Describe the soft tissue changes affecting the gingiva.
4. In a similar case, you take a bitewing radiograph 68.10B. Describe the radiographic findings.
5. Is this condition shown in the clinical photograph and radiograph normal, a variation of normal, or disease? And do you expect this condition to be symptomatic?
6. After reviewing the radiograph, does it appear that the alveolar bone is affected by this condition?
7. Which of the following is the most likely diagnosis?
 - A. Amalgam tattoo
 - B. Blue nevus
 - C. Intramucosal nevus
 - D. Melanoma
8. Why is taking a radiograph and interpretation of the film important in this case?



SECTION 10

Intraoral Findings by Surface Change

Dental Objectives:

- Define clinical entities that appear as nodules, papulonodules, vesicles, bullae, and ulcerative lesions of the oral cavity.
- Recognize the causes and clinical features of these conditions.
- Use the diagnostic process to distinguish similar-appearing nodules, papulonodules, and vesiculobullous and ulcerative lesions of the oral cavity.
- Recommend appropriate treatment options for these conditions.

Dental Hygiene Objectives:

- Define and use diagnostic terms that describe intraoral findings by surface change.
- In the clinical setting, document the characteristics of oral conditions discussed in this section in terms of:
 - a. Anatomic location
 - b. Color
 - c. Border
 - d. Configuration
 - e. Type
- Identify conditions discussed in this section that (1) require the attention of the dentist and/or (2) affect the delivery of periodontal debridement and oral hygiene measures.

In the figure legends, *†‡ denotes the same patient.

NODULES

Retrocuspid Papilla (Figs. 69.1 and 69.2) Not all persons have a **retrocuspid papilla**. This particular growth appears as a firm, round, fibroepithelial papule about 1 to 4 mm in diameter. It is located on the lingual surface of the attached gingiva of the mandibular cuspids just below or a few millimeters below the marginal gingiva. The surface mucosa is pink, soft, and smooth. Rarely, the structures may be pedunculated and the stalk can be lifted off the gingiva by a periodontal probe. The retrocuspid papilla is a variation of normal and is frequently found bilaterally. Some authorities state that it is a developmental anomaly that represents a variant form of fibroma. The retrocuspid papilla seems to be present in most children but regresses with maturity; thus, the incidence and size decrease with increasing age. The retrocuspid papilla has no sex predilection, and no treatment is necessary unless interference with a removable prosthesis is anticipated.

Oral Lymphoepithelial Cyst (Figs. 69.3 and 69.4) The **oral lymphoepithelial cyst** usually presents as a small, movable, painless swelling that is characterized by a well-circumscribed, soft, doughy mass. It is an encapsulated dermal or submucosal papule that arises from epithelium entrapped in lymphoid tissue that has undergone cystic transformation. The cyst is benign and asymptomatic, but may enlarge and spontaneously drain. Most appear in children and young adults. No sex predilection has been demonstrated.

Common sites for the oral lymphoepithelial cyst are the floor of the mouth, lingual frenum, ventral tongue, posterior lateral border of the tongue, and, rarely the soft palate. These small swellings rarely exceeding 1 cm in diameter that are characteristically yellow when superficial and pink when deeper. Palpation yields a slightly moveable nodule. When located in the anterior floor, the lesion may resemble a mucus-retention cyst. Infrequently, multiple lymphoepithelial cysts can be found.

When the lymphoepithelial cyst is derived from degenerative tissue of the second branchial arch, it is referred to as a **cervical lymphoepithelial cyst** or a **branchial (cleft) cyst**. This cyst appears on the lateral aspect of the neck just anterior and deep to the superior third of the sternocleidomastoid muscle near the angle of the mandible. The cyst may occur in the proximity of the parotid gland. The extraoral lymphoepithelial cyst is a well-circumscribed, soft, fluctuant mass that is rubbery to the touch. It may enlarge to 1 or 2 cm and can drain externally.

Histologic examination shows that lymphoepithelial cysts are usually lined with stratified squamous epithelium; occasionally, pseudostratified, columnar, or cuboidal epithelium is found. Surrounding the cystic epithelial lining is a fibrous connective tissue wall that contains dark-staining lymphoid aggregates with prominent germinal centers. The luminal fluid is yellow and viscous because of a cheesy keratinaceous content. Excisional biopsy should be performed to provide histologic confirmation. Lymphoepithelial cysts rarely recur.

Torus, Exostosis, and Osteoma (Figs. 69.5–69.8; Figs. 33.1 and 33.2) Tori, exostoses, and peripheral osteomas are readily recognizable, bony hard nodules that appear histologically identical. The term used depends on location, appearance, and systemic associations.

Tori are bony protuberances of the jaws, localized to the palatal midline or lingual surface of the mandible in the canine-premolar-molar area. They are the most common intraoral lesion. Women are more frequently affected. Tori are hard growths with smooth, rounded contours, normal-appearing or slightly pale mucosa, and a sessile base. They are often have a lobulated surface. Internally they are composed of dense cortical bone with an occasional central core of spongy bone. Hereditary factors contribute to the development of tori.

Exostoses are bony outgrowths in alternate locations of tori. The facial aspect of the maxillary and mandibular alveolar ridge are common sites. Infrequently, the palatal alveolar ridge adjacent to maxillary molars is affected. Most exostoses are multiple hard nodules that demonstrate creaselike folds between distinct nodules. The surface mucosa is firm, taut, and white to pale pink.

Tori and exostoses tend to increase slowly in size with advancing age but remain asymptomatic unless traumatized. After a traumatic incident, patients may be concerned about neoplasia and state that the bony mass is enlarging or that it was not present before the injury. Removal is generally unnecessary unless prompted by cosmetic, prosthodontic, psychological, or traumatic considerations.

Osteomas are benign neoplastic growths that are distinct from the developmental lesions (tori and exostoses) because osteomas have more growth potential, tend to be larger, and may rarely be confined to soft tissue. Almost all osteomas occur in the bones limited to the face and head. Two types are described based on surface of bone from which they arise. Those that arise on the outer surface of bone are known as **periosteal osteomas** (Figs. 33.2 and 33.4), whereas those arising within medullary bone are **endosteal osteomas**. Both types appear as radiodense structures with well-defined, smooth, and rounded borders. The presence of more than one osteoma dictates that a radiographic examination be performed for multiple impacted supernumerary teeth and odontomas, which may indicate the presence of **Gardner syndrome** (Figs. 20.7 and 20.8). This inherited condition, associated with a gene abnormality on chromosome 5, is characterized by osteomas, dermal cysts, multiple impacted and supernumerary teeth, odontomas, abnormal retinal pigmentation, and intestinal polyposis. The bowel polyps have a high propensity for malignant transformation. Most patients with Gardner syndrome demonstrate malignant polyposis by age 40; thus, all patients with this condition require close medical management. In addition, there is an increased risk of thyroid carcinoma in these patients.



Fig. 69.1. Retrocuspid papilla. Usual pink nodule appearance.

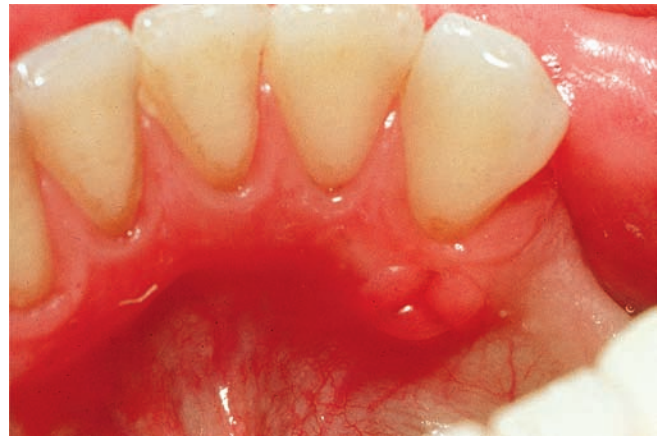


Fig. 69.2. Retrocuspid papilla: with unusual clefting.

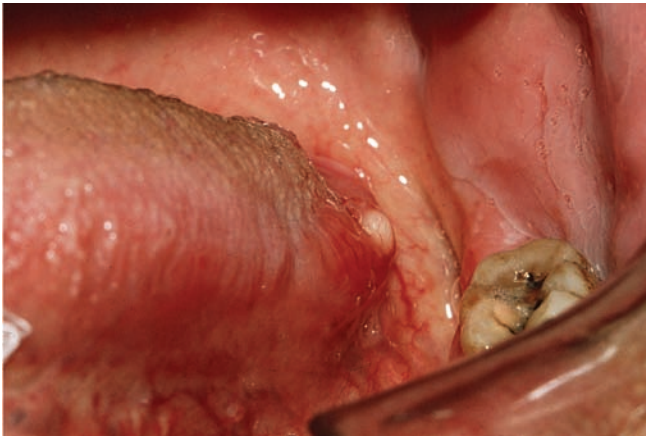


Fig. 69.3. Oral lymphoepithelial cyst: yellow, lateral tongue.

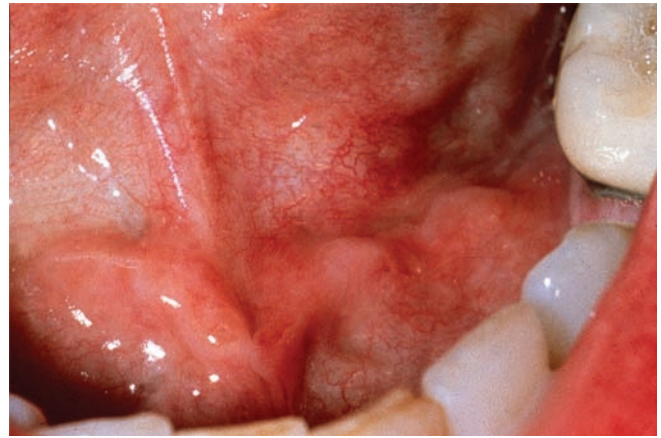


Fig. 69.4. Oral lymphoepithelial cyst: in floor of mouth.



Fig. 69.5. Mandibular tori: lobulated, bilaterally symmetric.



Fig. 69.6. Exostoses: palatal location.



Fig. 69.7. Exostoses: maxilla and mandible.



Fig. 69.8. Osteoma: enlarging, lingual to mandibular molars.

NODULES

Irritation Fibroma (Fig. 70.1) The **irritation fibroma** is one of the most common benign lesions of the oral cavity. It is a reactive hyperplasia that arises in response to a chronic irritant; thus, this lesion is not a true neoplasm as the term implies. True intraoral neoplastic fibromas are rare (see below). The irritation fibroma appears as a well-defined, pale pink papule that slowly enlarges to form a nodule. It is smooth, symmetrically round, firm, and painless. Infrequently, a white, roughened, or ulcerated surface is present because of repeated trauma. Most are sessile growths that arise on the buccal mucosa, labial mucosa, gingiva, or tongue. Histologic examination shows an interlacing mass of dense collagenous tissue covered by thinned epithelium. Fibromas are treated by removing the source of the irritation together with surgical excision. Fibromas occur mostly in adults. They recur infrequently if treated properly. Multiple intraoral (angio)fibromas, which resemble fibromas, are associated with **tuberous sclerosis**, an autosomal dominant disorder characterized by seizures, mental deficiency, and angiofibromas of the face.

Peripheral Odontogenic Fibroma The **peripheral odontogenic fibroma** is clinically similar to the irritation fibroma but characterized by its unique location and tissue of origin—arising from cells of the periodontal ligament. Accordingly, it is generally found in the region of the interdental papilla. An example is shown in Figure 40.7.

Giant Cell Fibroma (Fig. 70.2) The **giant cell fibroma** is a painless, pink papule or nodule that has a sessile base and smooth or slightly pebbly surface. Unlike the irritation fibroma, evidence of chronic irritation is generally lacking. The giant cell fibroma has many large, polynucleated stellate-shaped fibroblasts scattered among a loosely arranged vascular connective tissue; the irritation fibroma does not. Most giant cell fibromas occur before 35 years of age on the mandibular gingiva, tongue, or palate. Excision is recommended, and recurrence is rare.

Lipoma (Fig. 70.3) The **lipoma** is a common benign dermal tumor, but a rare intraoral finding. This slow-growing neoplasm is composed of mature fat cells surrounded by a thin, fibrous, connective tissue wall. Adults older than 30 years of age are commonly affected, and no sex predilection exists. In the mouth, lipoma present as a painless, smooth-dome-shaped, slightly lobulated, or diffusely elevated nodule that is yellow to pale pink. Sometimes they are polypoid, pedunculated, or lobulated. Most occur on the buccal mucosa or vestibule. Less common sites include the tongue, floor of the mouth, and lip. The palate is rarely involved (Fig. 52.2). Palpation reveals a doughy soft, movable, and compressible submucosal mass. Therapy consists of surgical removal; recurrence is rare.

Fibrolipoma (Fig. 70.4) The fibrolipoma is a rare benign intraoral neoplasm of mixed connective tissue origin. It is a well-demarcated submucosal mass consisting of mature lobules of fat cells intermixed with a significant fibrous connective tissue component. Clinical examination shows a blend of a fibroma and lipoma. It is generally found on labial and buccal mucosa as a nonindurated, movable, painless, and soft or firm lesion, depending on the fat-to-collagen content. They grow slowly but can obtain several centimeters in diameter.

Traumatic Neuroma (Figs. 70.5 and 70.6) A neuroma is a benign tumor of neural tissue. It arises *de novo* or as a result of trauma (amputation or traumatic neuroma). The **traumatic neuroma** results from a hyperplastic response to nerve damage after severance of a large nerve fiber. In the mouth, the traumatic neuroma is frequently encountered in the mandibular mucobuccal fold in the region adjacent to the mental foramen. It also arises facial to the mandibular incisors, lingual to the retromolar pad, and in ventral tongue. The size of the lesion depends on the degree of insult and hyperplastic response.

Traumatic neuromas are usually small nodules, measuring <0.5 cm in diameter. Visualization may be difficult if the lesion is located deep below normal oral mucosa. Neuromas are painful when palpated. Pressure applied to the neuroma elicits a response often described as an electric shock. Multiple neuromas discovered on the lips, tongue, or palate may indicate **multiple endocrine neoplasia type III**, also known as MEN 2b—an autosomal dominant condition characterized by numerous mucosal neuromas, marfanoid body type (thin elongated limbs), and endocrine neoplasms. Treatment of the traumatic neuroma is surgical excision or intralesional injection with corticosteroids. Excision may further damage the nerve and lead to recurrence.

Neurofibroma (Figs. 70.7 and 70.8) **Neurofibromas** are benign tumors resulting from proliferation of peripheral nerve components: the Schwann cells and perineural fibroblasts. They commonly appear as sessile, firm, pink nodules. They may be solitary or multiple. Solitary nodules are rare. More commonly, multiple, slowly enlarging neurofibromas are encountered with **neurofibromatosis** (von Recklinghausen disease; see “Swellings of the Face” [Fig. 55.6]).

Intraoral neurofibromas usually appear on buccal mucosa, tongue, and lips, and less commonly within bone. Most soft tissue neurofibromas are asymptomatic, but those within deeper tissues or bone may produce pain and paresthesia. Large neurofibromas can be soft and baggy, or firm and physically deforming. Within bone, they can expand the inferior alveolar canal. Solitary neurofibromas have no tendency for malignant transformation, but neurofibromatosis does.



Fig. 70.1. Irritation fibroma: on buccal mucosa.



Fig. 70.2. Giant cell fibroma: on dorsum of tongue.



Fig. 70.3. Lipoma: on lateral margin of tongue.



Fig. 70.4. Fibrolipoma: on labial mucosa.



Fig. 70.5. Traumatic neuroma: near midline and a papilloma.



Fig. 70.6. Neuromas of multiple endocrine neoplasia.



Fig. 70.7. Neurofibromatosis: multiple tumors, café-au-lait spot.



Fig. 70.8. Neurofibroma: nodule on lateral tongue.

PAPULONODULES

Oral Squamous Papilloma (Papilloma) (Figs. 71.1 and 71.2) **Papillomas** are the most common benign epithelial neoplasm of the oral cavity. They appear as small, pink-white, painless, exophytic masses that are usually <1 cm in diameter. The surface of the papule may be smooth, pink, and pebbly (vegetative) or have numerous small fingerlike projections. The base is pedunculated and well delineated. Intraoral lesions are typically soft, whereas keratin-producing lesions are rough or scaly. Lesions are generally solitary, but multiple lesions are occasionally seen. Human papillomavirus (HPV) types 6 and 11 have been detected in more than 50% of papillomas examined and are generally accepted as the cause of this disease.

The mean age of occurrence of papilloma is 35 years; both sexes are affected equally. Most occur on the palate, uvula, tongue, frenum, lips, buccal mucosa, and gingiva. Other HPV-induced lesions, such as the condyloma acuminatum, focal epithelial hyperplasia (Heck disease), and verruca vulgaris, share similar clinical features but are microscopically distinct. Histologic features include fingerlike epithelial projections and a fibrovascular core. Treatment is complete excision, including the base. Recurrence is rare. If left untreated, they can enlarge, spread to other sites, and serve to transmit HPV to other persons. Malignant transformation does not occur.

Verruca Vulgaris (Fig. 71.3) **Verruca vulgaris** is the common skin *wart* that seldom occurs intraorally. The etiologic agents are HPV types 2, 4, 6, 11, and 40. Virally induced cellular changes result in the characteristic clinical findings. The lesional surface is typically rough and raised, with white fingerlike projections. The whiteness of intraoral verrucae varies, depending on the amount of surface keratinization. Pink areas are not unusual at the lesional base. Verrucae are contagious and commonly located on the skin, lip, labial/buccal mucosa, tongue, and attached gingiva. The base of the lesion is broad, but the size is usually <1 cm. On clinical examination, it may appear identical to a papilloma, although the clefts are more shallow and the mass more sessile. Viral inclusion bodies are frequently seen on histologic examination. Patients with skin verrucae are more likely to have oral lesions as a result of autoinoculation. A lesion may sometimes regress spontaneously. If not, treatment is effective by excision, laser removal, cryosurgery, or chemical applications. Recurrence is possible.

Focal Epithelial Hyperplasia (Heck Disease) (Fig 71.4) **Focal epithelial hyperplasia** is a virus-induced disease associated with multiple asymptomatic, papulonodular growths of the oral mucosa, particularly the tongue and labial and buccal mucosa. It was first described in Native Americans and Eskimos but now has been reported in many populations. The etiologic agents are HPV types 13 and 32. The virus is transmitted during kissing. Virus replication in epithelial cells produces

soft papular growths in children and teenagers. Lesions are initially small, discrete, flat papules that are pink or whitish pink. Later lesions enlarge, become papillary or cobblestoned, and can coalesce. Some lesions undergo spontaneous regression; surgical excision can be performed for those that do not regress.

Condyloma Acuminatum (Venereal Wart) (Figs. 71.5 and 71.6) The **condyloma acuminatum** is a transmissible papillomatous growth that occurs orally much less frequently than the papilloma. The warm, moist, intertriginous areas of the anogenital skin and mucosa are frequent sites of growth. In more than 85% of condyloma acuminata, HPV types 6 and 11 DNA is present in the epithelium. The virus is transmitted by sexual and oral contact.

Condyloma acuminata are usually small, pink to dirty gray, solitary or multiple growths. The surface is often pebbly, resembling a cauliflower, but may be flat. The base is sessile, and the borders are raised and rounded. When multiple lesions are present, proliferation of adjacent condylomas can form extensive clusters that may appear as a single mass. Any oral mucosal surface may be affected, but the labial mucosa is the most common site. Other sites include the tongue, lingual frenum, gingiva, and soft palate. Histologic examination shows parakeratosis, cryptic invagination of cornified cells, and koilocytosis. Wide excision or antiviral agents are used in treatment, as condylomata have a high rate of recurrence. Occasional oncogenic transformation of long-standing anogenital growths has been reported, but this is not the case with oral lesions.

Lymphangioma (Figs. 71.7 and 71.8) A **lymphangioma** is a collection of lymph vessels (benign hamartomas) that are overgrown and clumped together. Lymphangiomas develop early in life with no sex predilection. They may occur on the skin or mucous membrane. In the oral cavity, they occur commonly in the dorsal and lateral surface of the anterior portion of the tongue, lips, and labial mucosa.

Small superficial lymphangiomas have irregular papillary projections that resemble a papilloma. They are soft and compressible and vary from normal pink to whitish, slightly translucent, or blue. Deep-seated lesions cause diffuse enlargement and stretching of the surface mucosa. A diffuse lymphangioma in the tongue causes macroglossia; in the lips, it causes macrocheilia; and in the neck, it is called a **cystic hygroma**. Aspiration or diascopy is mandatory before surgical excision of a lymphangioma to prevent complications associated with the similar-appearing hemangioma. Patients with a large, diffuse lesion often require hospitalization to monitor postoperative edema and possible airway obstruction. Lymphangiomas do not undergo malignant change. Some lymphangiomas, especially congenital types, regress spontaneously during childhood.



Fig. 71.1. Oral squamous papilloma: on soft palate.



Fig. 71.2. Papilloma: with fingerlike projections.



Fig. 71.3. Verruca vulgaris: autoinoculation from fingers.



Fig. 71.4. Focal epithelial hyperplasia: multiple on labial mucosa.



Fig. 71.5. Condyloma acuminata: on ventral tongue.



Fig. 71.6. Condyloma acuminata: multiple on labial mucosa.



Fig. 71.7. Lymphangioma: causing macroglossia.*



Fig. 71.8. Lymphangioma: pebbly, pink-white in lip.*

VESICULOBULLOUS LESIONS

Primary Herpetic Gingivostomatitis (Figs. 72.1–72.3) Herpes simplex virus (HSV) types 1 and 2 belong to the family Herpesviridae, which includes eight viruses (cytomegalovirus, varicella-zoster virus, Epstein-Barr, and human herpes virus type 6, 7, and 8). Approximately 75% to 90% of the adult human population has been infected with HSV. Transmission occurs by contact of infected oral secretions with the mucosa or skin of a susceptible person. In most cases of primary infection, HSV-1 is the causative organism; however, HSV-2, which has a propensity to infect the skin below the waist, can cause herpetic gingivostomatitis by oral-genital or oral-oral contact.

The **primary HSV infection** may be trivial or fulminating. Subclinical signs that go unrecognized or may produce flulike symptoms are the norm. When symptomatic, the infection is called **primary herpetic gingivostomatitis**. It occurs most commonly in children younger than 10 years and second most commonly in young adults. The acute inflammatory response of the primary HSV infection usually follows a 2- to 10-day incubation period. Infected patients report fever, malaise, and irritability. Focal areas of the marginal gingiva initially become fiery red and edematous. The interdental papillae abruptly swell and bleed after minute trauma because of capillary fragility and increased permeability. Widespread inflammation of the marginal and attached gingiva develops, and small clusters of vesicles rapidly erupt throughout the mouth. The vesicles burst, forming yellowish ulcers that are individually circumscribed by a red halo. Coalescence of adjacent lesions forms large ulcers of the buccal mucosa, labial mucosa, gingiva, palate, tongue, and lips. Shallow erosions of the perioral skin and hemorrhagic crusts of the lips are characteristic. Headache, lymphadenopathy, and pharyngitis are common, especially in young adults infected with HSV-2.

Pain is a significant problem in patients with primary herpetic gingivostomatitis. Mastication and swallowing may be impaired, resulting in dehydration and subsequent elevation of temperature. Viral culturing, serum antibody levels, and results of cytologic testing are helpful in the diagnosis. Treatment is supportive and should include antiviral agents if given within the first few days of disease onset. Patients with a high temperature (exceeding 101°F) should receive nonaspirin antipyretic drugs and antibiotics.

Primary herpetic gingivostomatitis is a contagious disease that usually regresses spontaneously within 12 to 20 days without scarring. Complications associated with the primary infection include autoinoculation of other epidermal sites, producing keratoconjunctivitis (eye) and **herpetic whitlow** (finger); extensive epidermal infection in the atopic patient, which is called **Kaposi varicelliform eruption**; and disseminated infections in immunosuppressed patients. Immunity to HSV is relative, and patients previously infected with the virus may be reinfected with a different strain of HSV.

Recurrent Herpes Simplex Infection (Figs. 72.4–72.7) After the acute infection, HSV infects sensory nerve endings, migrates to regional sensory ganglia, and enters latency within infected neurons. Reactivation of virus and clinical recurrences develop in about 40% of persons who harbor latent virus. Recurrences are often precipitated by sunlight, heat, stress, trauma (dental procedures), or immunosuppression. Normal immune mechanisms are required to eliminate reactivated HSV. Asymptomatic shedding of virus into saliva in the absence of oral lesions occurs in 10% of the population on any given day.

Recurrent herpes simplex infection produces clusters of vesicles that ulcerate. The vesicles repeatedly develop at the same site, following the distribution of the infected nerve. Recurrences on the vermilion border of the lip (**recurrent herpes labialis**) are clinically more apparent than intraoral recurrences (**recurrent herpetic stomatitis**). The lesions of recurrent herpes labialis appear as a small cluster of vesicles that erupt, coalesce, ulcerate, form a scab, and heal without scarring. Spread to perioral skin is common, especially with use of greasy lip ointments that permit horizontal weeping of vesicular fluid. Contact of vesicular fluid at other epidermal sites can result in spread of the infection. In relatively healthy persons, recurrent herpetic stomatitis produces small ulcers with red halos that are limited to periosteal-bound, keratinized mucosa (i.e., the attached gingiva and hard palate). Recurrences on the buccal mucosa and tongue are infrequent unless the patient is immunosuppressed.

Most patients with recurrent herpes simplex report pain and tenderness of affected tissues. Prodromal neurogenic symptoms, such as tingling, throbbing, and burning, often precede the eruption of lesions by 6 to 24 hours. Sunscreens are effective in the prevention of lip and skin recurrences. Management also includes lysine dessicating agents and antiviral drugs (acyclovir, famciclovir, penciclovir, and valacyclovir). Patients should be informed that lesions are contagious and that saliva is contaminated with virus.

Herpangina (Fig. 72.8) Herpangina is a highly contagious but self-limiting infection involving the oral cavity that is caused by group A and sometimes group B coxsackie viruses, and echovirus. The disease is spread by infected saliva. It is seen mainly in children during the warmer months of summer. Young adults are occasionally affected. It produces a small number of light-gray papillary vesicles that rupture to form discrete, shallow ulcers. The ulcers have an erythematous border and are limited to the posterior oral cavity (tonsillar pillars, soft palate, uvula). Diffuse pharyngeal erythema, dysphagia, and sore throat are common features, as are fever, malaise, headache, lymphadenitis, abdominal pain, and vomiting. Convulsions rarely occur. Treatment is palliative, and spontaneous healing occurs within 1 to 2 weeks.



Fig. 72.1. Primary herpetic gingivostomatitis: in a 7-year-old.*



Fig. 72.2. Primary herpetic gingivostomatitis.*



Fig. 72.3. Primary herpetic gingivitis: a 27-year-old man.



Fig. 72.4. Recurrent herpes labialis: clustered vesicles.



Fig. 72.5. Recurrent herpes simplex: multiple gingival ulcers.



Fig. 72.6. Recurrent herpes simplex: on hard palate.



Fig. 72.7. Herpetic whitlow: caused by autoinoculation.



Fig. 72.8. Herpangina: multiple, soft palate ulcers and redness.

VESICULOBULLOUS LESIONS

Varicella (Chickenpox) (Figs. 73.1 and 73.2) Varicella and herpes zoster are caused by the same virus, varicella-zoster virus (VZV). **Varicella** (chickenpox) is the highly contagious primary infection, whereas **herpes zoster** is the recurrent neurodermal infection. Typically, children become infected with the virus during the late winter and spring months. After exposure to the virus and a 2- to 3-week incubation period, mild prodromal features appear.

Chickenpox is usually a mild childhood illness. Fever, malaise, and a distinctive red and very itchy rash on the face and trunk are the first recognizable signs of the disease. The pruritic rash spreads quickly to the neck and extremities and is followed shortly by the eruption of papules that form vesicles and pustules. On bursting, the pus-laden vesicles have the appearance of a dew-drop on a rose petal. The first and largest skin lesion is called the *herald spot*. It is often located on the face and, if scratched, may heal with scarring. Infected persons are contagious from 2 days before the rash appears until all lesions crust over.

Intraoral lesions of varicella are few and often go unnoticed. They appear as vesicular lesions that break down and form ulcers with an erythematous halo. The soft palate is the predominant site, followed by the buccal mucosa and mucobuccal fold. Anorexia, fever, chills, headache, nasopharyngitis, and muscle aches may accompany the condition. Complications such as pneumonitis and encephalitis are infrequent; however, about 100 people die each year in the United States as a result of complications. Vesicles typically crust over and resolve spontaneously within 7 to 10 days. Infection during pregnancy poses a significant risk to the fetus. A live-attenuated vaccine (Varivax) is recommended to be given to infants between 12 and 18 months to prevent the development of chickenpox.

Herpes Zoster (Shingles) (Figs. 73.3 and 73.4) **Herpes zoster** is the recurrent infection of chickenpox. Unknown factors associated with aging, cancer, and immunosuppression result in reactivation of dormant varicella virus from sensory ganglia and migration of virus along the affected sensory nerves. The prevalence increases with age, with up to 20% of the population being affected, usually after 50 years of age. Rarely, young adults or children are affected. Before eruption, prodromal signs of itching, tingling, burning, pain, or paresthesia occur. Lesions are characterized by acutely painful vesicular eruptions of the skin and mucosa that are unilaterally distributed along one dermatome and stop abruptly at the midline. Two areas are affected the most: (1) the trunk between vertebrae T3 and L2; and (2) the face along the ophthalmic division of the trigeminal nerve.

Cutaneous lesions of shingles begin as pruritic, erythematous macules that become vesicular and pustular eruptions. Crust formation occurs within 7 to 10 days and persists for several weeks. Pain is intense but usually dissipates after crusts fall off.

Typically, an intense prodromal pain develops 1 to 2 days before intraoral lesions erupt. Lesions develop as clusters of vesicles that ulcerate with an intense red, inflammatory border. Within several days, a yellowish surface slough forms, and zones of bleeding occur. Involvement of the mandibular branch of the trigeminal nerve results in ulcers of the lips, tongue, and buccal mucosa that extend to the midline. Involvement of the maxillary nerve branch typically produces unilateral palatal ulcerations that extend up to but not beyond the palatal raphe. Considerable malaise, fever, and distress accompany the disease, and in some cases, the teeth may become devitalized or bone necrosis can result.

Herpes zoster usually heals without scar formation in about 3 weeks. However, scarring can occur, and many patients may experience persistent pain after the lesions have faded. This condition, called **postherpetic neuralgia**, may continue for months before regressing. Immunosuppressed patients are particularly susceptible to shingles and have a high morbidity rate. Varicella-zoster virus infection that causes unilateral facial paralysis and involves the auditory nerves (ear eruptions) is known as **Ramsay Hunt syndrome**. The disease can be associated with **Reye syndrome** (high fever, cerebral edema, liver degeneration, high mortality) if salicylates are used to manage the disease in children. Antiviral agents, famciclovir (Famvir), and valacyclovir (Valtrex), are highly effective in blocking replication of varicella-zoster virus and should be used within 72 hours of onset of the disease. A vaccine is available to the elderly.

Hand-Foot-and-Mouth Disease (Figs. 73.5–73.8) **Hand-foot-and-mouth disease** is a mildly contagious disease caused by many Coxsackie A and B viruses. It usually affects children but may be seen in young adults, typically during spring and summer. As the name implies, it produces small ulcerative lesions in the mouth together with an erythematous rash on the skin of the hands, fingers, and soles of the feet. Rarely, the legs and lower trunk are involved. Multiple pinpoint vesicles that ulcerate and crust are characteristic. Patients may have several to more than 100 pinpoint lesions with distinctive erythematous halos.

Oral lesions of hand-foot-and-mouth disease are scattered mainly on the tongue, hard palate, and buccal and labial mucosa. In time, they coalesce to form large eroded areas. Unlike herpangina, the oropharynx is usually unaffected, and the total number of intraoral lesions ranges up to 30. Pain and flulike symptoms (fever, malaise, and lymphadenopathy) are common, along with cough, anorexia, vomiting, and diarrhea. The diagnosis can be made by viral culture and serum antibody studies. However, the classic distribution of lesions on the palms of hands, soles of feet, and oral mucosa is diagnostic in most instances. Healing occurs regardless of treatment in about 10 days.



Fig. 73.1. Varicella (chickenpox): herald spot lateral to eye.



Fig. 73.2. Varicella (chickenpox): vesicle near midline.



Fig. 73.3. Herpes zoster (shingles): mandibular eruption.*



Fig. 73.4. Herpes zoster (shingles): painful oral lesions.*



Fig. 73.5. Hand-foot-and-mouth disease: pinpoint ulcer.†



Fig. 73.6. Hand-foot-and-mouth disease: foot ulcer.†



Fig. 73.7. Hand-foot-and-mouth disease: labial ulcers.‡



Fig. 73.8. Hand-foot-and-mouth disease: coalescing and painful.‡

VESICULOBULLOUS LESIONS

Allergic Reactions (Figs. 74.1–74.8) **Allergy** is an abnormal or hypersensitive response of the immune system to a usually harmless substance in the environment, such as mold or pollen. These reactions are acquired by repeated exposure to an allergen, which results in inappropriate tissue damage from antigen–antibody reactions. Manifestations may be generalized or localized and occur at any age. A genetic predisposition is common.

Hypersensitivity reactions are classified into several types according to the following factors: the speed with which the symptoms occur (immediate or delayed); clinical appearance; and cellular and tissue response (**type I**: IgE-mediated immediate hypersensitivity; **type II**: antibody-dependent cytotoxic hypersensitivity; **type III**: complex-mediated hypersensitivity; and **type IV**: cell-mediated, or delayed, hypersensitivity). Reactions of dental significance include immediate type I reactions (anaphylactic shock, urticaria, angioneurotic edema, allergic stomatitis) and delayed type IV reactions (contact allergy).

Localized Anaphylaxis (Figs. 74.1–74.2) **Localized anaphylaxis** is an immediate allergic response mediated by IgE and histamine that occurs within minutes of exposure to an antigen. The localized condition produces vasodilation and increased permeability of superficial blood vessels, tissue swelling, and pruritus. The localized form manifests as wheals, urticaria, or hives that arise after the ingestion of such foods as shellfish, citrus fruits, peanuts, chocolate, or systemically administered drugs.

Generalized Anaphylaxis A rapid and potentially life-threatening allergic (type I hypersensitivity) reaction is **generalized anaphylaxis**. This sudden systemic reaction causes constriction of the airways, resulting in difficulty breathing or even unconsciousness and death. It results from an antigen–antibody (IgE) interaction that results in mast cell degranulation and the release of vasoactive amines and mediators such as histamine. In severe cases, a generalized increase in vascular permeability and smooth-muscle contraction causes urticaria, dyspnea, hypotension, airway edema, and vascular collapse. Mild localized immediate hypersensitivity reactions are treated with antihistamines. Epinephrine 1:1,000 (0.3–0.5 mL, subcutaneously) and airway management is required to effectively manage severe reactions. Treatment should always target elimination of the causative allergen.

Allergic Stomatitis (Fig. 74.3) **Allergic stomatitis**, also called **allergic mucositis**, is an oral type I hypersensitivity reaction to a systemically administered drug or food. The oral manifestations (eruptions) occur rapidly but are varied in appearance. Some resemble erythema multiforme or lichen planus. In the mouth, a dry, glistening, red area is usually apparent. Focal white areas may be adjacent. Multiple vesicles form that break down and produce fibrin-covered ulcers. The ulcers have red, inflammatory borders and are painful or

burn. The reaction may be limited to the buccal/labial mucosa, gingiva, lips, or tongue or may involve the entire oral cavity. Concurrent dermal lesions are possible. Treatment requires withdrawal of the allergen and administration of antihistamines.

Angioedema (see Figs. 50.1, 50.2, and 56.1) **Angioedema** is a swelling within the tissues usually occurring around the eyes and lips. Angioedema results from a hypersensitivity reaction brought about by histamine-induced vascular leakage. Hereditary and acquired forms exist. The hereditary form, caused by a activation of complement, is more serious. Both forms are characterized by swelling that rapidly appears and lasts for 24 to 36 hours. Sensations of warmth, tenseness, and itchiness are concurrent. See “Swellings of the Lip” (Fig. 50.1) and “Conditions Peculiar to the Face” (Fig. 56.1) for a full discussion.

Delayed Hypersensitivity (Figs. 74.4 and 74.5) **Delayed hypersensitivity** or type IV hypersensitivity is a response of the immune system to a locally or systemically introduced allergen that usually develops slowly and reaches its maximum 24 to 48 hours after antigenic exposure. Topically applied allergens, such as latex gloves or chemical disinfectants, may produce a delayed hypersensitivity response evident as itchy, erythematous skin lesions (contact dermatitis) that eventually become inflamed and ulcerated at the site of contact. Delayed hypersensitivity is treated with corticosteroids and avoidance of the allergen.

Contact Stomatitis (Figs. 74.6 and 74.7) **Contact stomatitis** is a form of delayed hypersensitivity that is due to T-cell attack. It produces erythema at the site of contact with the allergen. Reactions to lipstick or sunscreens may cause the lips to burn and appear red, swollen, fissured, or dry. Toothpastes, mouthwashes, antibiotic lozenges, topical anesthetics, and eugenol preparations can cause diffuse reactions. Intraoral lesions typically appear on the alveolar mucosa, dorsum of the tongue, or palate as erythematous ulcers that are covered by a gray-white pseudomembrane. Cast alloy restorations and partial denture frameworks that contain heavy metals (cobalt, mercury, nickel, or silver) can induce delayed hypersensitivity reactions of the mucosa adjacent to the restored area. The area is usually red and ulcerated, and often burns. Allergy to the free monomer in dentures, once thought to be common, is a rare occurrence.

Plasma Cell Gingivitis (Fig. 74.8) **Plasma cell gingivitis** affects the gingiva, producing diffusely edematous and fiery red gingiva because of the flavoring ingredients (such as cinnamon) in some toothpastes and chewing gums. The lips and commissures are frequently involved, resulting in cheilitis. Microscopy shows tissue infiltrated with plasma cells, a type of differentiated B cell that produces antibodies. Withdrawal of the causative agent is curative.



Fig. 74.1. Immediate (type I) hypersensitivity: facial wheal.

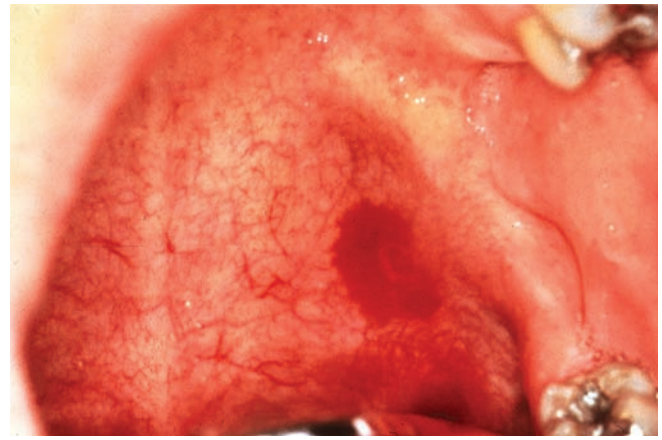


Fig. 74.2. Immediate (type I) hypersensitivity: bee sting.



Fig. 74.3. Immediate (type I) hypersensitivity: penicillin.

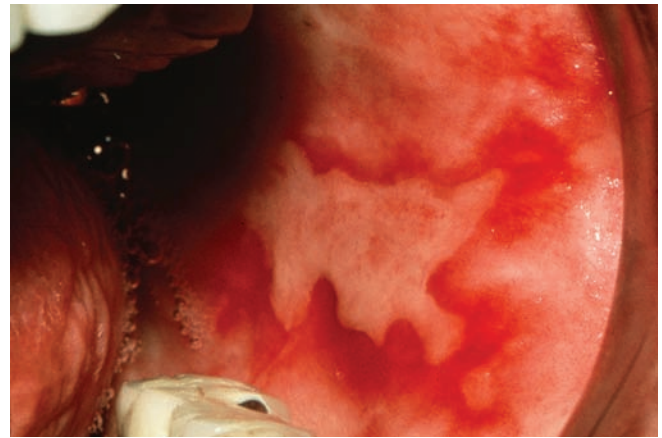


Fig. 74.4. Delayed (type IV) hypersensitivity: from thiazide.



Fig. 74.5. Delayed (type IV) hypersensitivity: latex allergy.

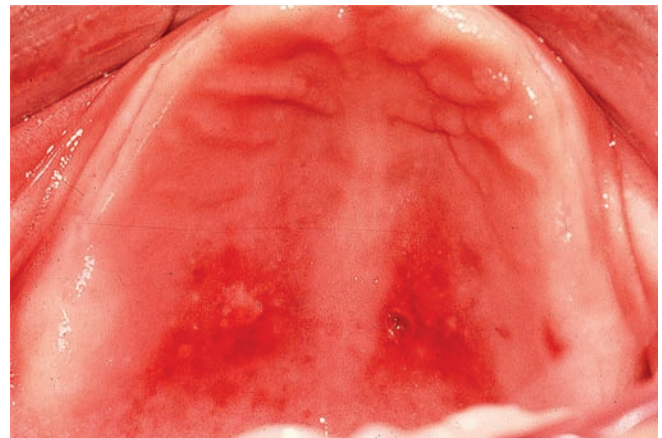


Fig. 74.6. Delayed (type IV) hypersensitivity: benzocaine.



Fig. 74.7. Delayed (type IV) hypersensitivity: alloy contact.



Fig. 74.8. Plasma cell gingivitis: type IV hypersensitivity.

VESICULOBULLOUS LESIONS

Erythema Multiforme (Figs. 75.1–75.4) **Erythema multiforme** is an acute inflammatory disease of varied skin and/or mucous membranes involvement characterized by red target-shaped macules and ulcers caused by hypersensitivity to a drug, microbe, or other allergen. It commonly affects young adults, particularly men, but may affect children and the elderly. Low-grade fever, malaise, and headache typically precede the emergence of lesions by 3 to 7 days. Most cases are the result of an immunological response to drug administration, especially drugs containing sulfa (antibiotics or hypoglycemic agents) or barbiturates. Other cases are precipitated by radiation, infections with herpes simplex virus or *Mycoplasma pneumoniae*, or an unidentified allergen. Immune complexes form that settle in small blood vessels, resulting in perivascular inflammation and necrosis of the epithelium.

Erythema multiforme can be classified into four types according to its spectrum of clinical presentations.

Oral Erythema Multiforme (Fig. 75.1) Oral erythema multiforme is the minimal manifestation of erythema multiforme. It is usually limited to the gingiva, but eruptions may also affect the tongue, lips, or palate. A history of infection or drug therapy is common. Constitutional symptoms such as anorexia, malaise, and low-grade fever may or may not be present. As the name multiforme (*many forms*) suggests, the lesions have a *varied appearance*. Affected gingiva appears fiery red, similar to the appearance of desquamative gingivitis; mucosal surfaces of the tongue and lips often show diffuse red patches with zones of ulceration. The borders of lesions are irregular (wavy) and red but seldom hemorrhagic, as is seen in pemphigoid and pemphigus.

Erythema Multiforme (Figs. 75.2–75.4) The hallmarks of classic erythema multiforme are the red-white, concentric, ringlike macules termed *target*, *bull's-eye*, or *iris lesions* that rapidly appear on the extremities (arms, legs, knees, and palms of the hands). The trunk of the body is classically exempt from lesions, except in the most severe cases. The skin lesions are initially small, dusky red, circular macules that vary in size from 0.5 to 2.0 cm in diameter. The macules then enlarge and develop a pale white or central clear area. Shortly thereafter, the lesions slightly elevate and become urticarial plaques that form vesicles and bullae. The vesicles may go unnoticed until they rupture and become confluent, forming large, raw, and shallow ulcers with erythematous borders. A necrotic slough and a yellowish fibrin pseudomembrane typically cover the ulcers.

In the mouth, red macular areas, multiple ulcerations, and erosions with a gray-white fibrinous surface may be seen. These are generally limited to the buccal mucosa, labial mucosa, or tongue or involve all of those areas. The gingiva and palate are sometimes involved. Dark red-brown, hemorrhagic (bloody) crusts are characteristically present on the lips, which helps establish the diag-

nosis. Lesions are usually short-lived and last about 2 weeks. Erythema multiforme rarely persists for more than 1 month. About 20% of patients experience recurrent episodes and chronic disease.

Pain is the most common symptom. Oral hygiene may be neglected, resulting in secondary bacterial infection. Treatment consists of topical palliative rinses and, in some instances, systemic corticosteroids. If a viral infection precedes the episode, antiviral therapy is provided. Evidence of a drug trigger dictates that drug administration be stopped. Complications resulting from erythema multiforme are uncommon unless the disease progresses to its major form, Stevens-Johnson syndrome.

Stevens-Johnson Syndrome (Erythema Multiforme Major) (Figs. 75.5–75.7) A severe form of erythema multiforme is termed **erythema multiforme major** or **Stevens-Johnson syndrome**, named for the two investigators who first described the appearance of the disease in the early 1920s. It frequently affects children and young adults, predominantly men. Stevens-Johnson syndrome is typically triggered by a drug and is characterized by widespread involvement of cutaneous and stomatologic structures, and constitutional signs such as fever, malaise, headache, chest pain, diarrhea, vomiting, and arthralgia.

The classic clinical triad of Stevens-Johnson syndrome consists of *eye lesions (conjunctivitis)*, *genital lesions (balanitis, vulvovaginitis)*, and *stomatitis*. Other features include the characteristic target skin lesions on the face, chest, and abdomen that later develop into painful weeping bullae. Like erythema multiforme, the gingiva is less commonly affected by desquamating bullae than is the nonkeratinized mucosa. Extensive ulcerative and hemorrhagic lesions of the lips and denuded areas of oral mucosa are intensely painful and usually prevent affected patients from eating and swallowing. Inadequate nutritional intake, dehydration, and debilitation are common sequelae that necessitate rehydration and hospitalization. Healing takes about 6 weeks.

Toxic Epidermal Necrolysis (Fig. 75.8) **Toxic epidermal necrolysis** is the most severe form of erythema multiforme. Its occurrence is rare, and most cases are associated with drug therapy. Unlike other forms of erythema multiforme, older persons are most commonly affected, especially women. The condition primarily affects the skin, eyes, and oral mucosa; the skin manifestations are especially severe. Large areas of the skin form coalescing bullae that slough, leaving huge areas of denuded skin. Management is similar to that of a burn patient. Significant morbidity will occur if supportive therapy is not provided. Treatment consists of intravenous fluid, nutritional therapy, corticosteroids, anesthetic and antiseptic rinses, and prevention of secondary infection with antibiotics. Affected areas take weeks to heal, and permanent eye damage is a frequent outcome. Both Stevens-Johnson syndrome and toxic epidermal necrolysis have been fatal.



Fig. 75.1. Oral erythema multiforme: gingival involvement.



Fig. 75.2. Erythema multiforme: classic target skin lesions.



Fig. 75.3. Erythema multiforme: hemorrhagic lip crusts.

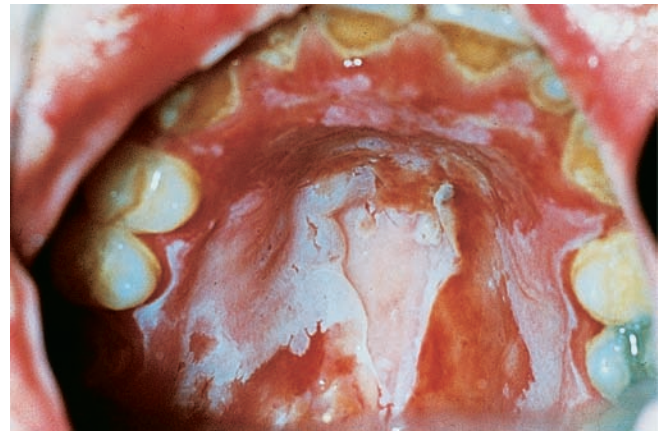


Fig. 75.4. Erythema multiforme: erythema and ulceration.



Fig. 75.5. Stevens-Johnson syndrome: eye, skin involvement.



Fig. 75.6. Stevens-Johnson syndrome: blood-crusts lips.



Fig. 75.7. Stevens-Johnson syndrome: genital involvement.*



Fig. 75.8. Toxic epidermal necrolysis: life threatening.

VESICULOBULLOUS LESIONS

Pemphigus Vulgaris (Figs. 76.1–76.4) **Pemphigus** is a potentially fatal, autoimmune skin disease that causes often large bullae (blisters) and erosions of the skin and mucous membranes. Four types have been described: *vulgaris* and *vegetans*, which have intraoral manifestations, and *foliaceus* and *erythematosus*, which generally do not. **Pemphigus vulgaris**, the most common intraoral type, develops more often in women between 40 and 60 years of age, and in light-pigmented patients of Jewish or Mediterranean origin. It is infrequent in children and the elderly. Acute and chronic forms exist; the slow chronic form is more common.

Pemphigus vulgaris is caused by autoantibody destruction of the adhesion proteins (desmogleins) of epithelium that compose the desmosome. Desmosomes are the intercellular glue-like substance that hold epithelial cells together. Destruction of desmogleins (type 3 and 1) causes cell-to-cell separation (acantholysis), particularly the basal cell layer from the overlying stratum spinosum. Events that induce autoantibody production (antidesmoglein 3 IgG) are unknown, but occasionally are induced by drugs. Destruction of the attachment proteins produces intraepithelial splitting or blisters (bullae) that rupture, quickly leaving painful erosions of skin and oral mucosa. Lesions develop rapidly and form weeping bullae or clear gelatinous plaques (a collapsed bulla). The bullae are extremely fragile and rapidly disintegrate, bleed, and crust. They tend to recur and spread. Light lateral pressure applied to a bulla causes the blister to enlarge by extension (the **Nikolsky sign**). A characteristic mucosal finding is the appearance of a whitish superficial covering, which is the roof of a collapsed bulla that can be easily stripped away.

Pemphigus may appear as sloughing or folded epithelium, an ulcer, or multiple, large irregular ulcers. Most often, the buccal mucosa, gingiva, palate, floor of the mouth, and lips are involved. Less frequently, the tongue and oropharynx are affected. Individual lesions have circular or serpiginous borders, whereas extensive erosions of the buccal mucosa appear red and raw and have diffuse irregular borders. New eruptions may superimpose over healing lesions so periods of remission are absent. Feter oris (bad odor), hemorrhagic lip crusts, and severe pain are characteristic.

The diagnosis of pemphigus is made by positive Nikolsky sign, biopsy, and immunofluorescent staining. Early recognition of oral lesions, which precede skin involvement by several months in more than half the cases, is important because early diagnosis greatly enhances the initiation of treatment and the prognosis. Remission and control are gained with use of corticosteroid and immunosuppressive agents. Dehydration and septicemia are potentially fatal complications. A small percentage of cases are associated with an underlying neoplasm (i.e., **paraneoplastic pemphigus**).

Pemphigoid (Bullous and Cicatricial) (Figs. 76.5–76.8) **Pemphigoid** is a chronic, self-limiting, autoimmune disease of older adults that involves the oral cavity in about 90% of cases. It is more common intraorally than pemphigus, but associated with much less morbidity and mortality. Pemphigoid is caused by separation of the epithelium from the basement membrane. Two types of pemphigoid (**bullous** and **cicatricial**) and several subgroups exist. They produce identical oral lesions and are distinguished by clinical and immunohistologic features.

In **bullous pemphigoid**, the less common of the two, skin lesions predominate over oral lesions. Skin folds of the axilla, inguinal, and abdominal regions are commonly affected. **Cicatricial pemphigoid**, also called **benign mucous membrane pemphigoid**, is more common and is characterized by lesions predominantly of mucous membranes, particularly the ocular and oral mucosa. It occurs twice as frequently in women as men and usually develops after 50 years of age. There is no racial predilection.

Pemphigoid occurs when autoantibodies (IgG, IgG4, IgM, or IgA) bind and destroy the anchoring filament complex of the basement membrane at the dermal-epidermal junction. Bullous pemphigoid targets the 230-kilodalton basement protein (BP230) and collagen XII at sites within the upper layer of the basement membrane (lamina lucida). In contrast, cicatricial pemphigoid targets proteins, laminin 5 or BP180, near the lower layer of the basement membrane (lamina densa). This leads to detached epithelium at the level of the lamina lucida, thus exposing the underlying connective tissue. Immunofluorescence staining reveals linear deposits of immunoglobulin (Ig)G and C3 along the basement membrane.

Pemphigoid skin lesions usually precede oral lesions, tend to be desquamative and localized, and heal spontaneously. The lips are rarely affected. Intraoral bullae are usually small, yellow, or hemorrhagic blebs. They form slowly and favor the palate, gingiva, and buccal mucosa. Because pemphigoid bullae result from subepithelial separation (Nikolsky positive), they are thicker walled, less fragile, and longer lasting than those of pemphigus. Rupture leads to patchy ulcers that coalesce. The ulcers are symmetric, curvilinear, and surrounded by a red border. When limited to the gingiva, the clinical term **desquamative gingivitis** has been used to describe the bright red, burning, and denuded tissue. Desquamative gingivitis is a descriptive term and may represent several clinically similar vesiculobullous conditions.

Cicatricial pemphigoid may affect the anal, vaginal, and pharyngeal mucosa, but the most severe complication is ocular involvement, producing conjunctivitis, occasional bullae, clouding of the cornea, and corneal healing marked by scarring and possibly blindness. Although the condition is rarely fatal, close follow-up is suggested because rare reports of carcinoma of the rectum and uterus have been associated with pemphigoid. Moderate doses of dapsone and corticosteroids, alone or in conjunction with immunosuppressive agents such as azathioprine, have provided effective management.



Fig. 76.1. Pemphigus vulgaris: lip and nose crusts.



Fig. 76.2. Pemphigus vulgaris: hemorrhagic lip crusts.



Fig. 76.3. Pemphigus vulgaris: rare, intact bullae on mucosa.*



Fig. 76.4. Pemphigus vulgaris: erythema below ruptured bullae.*

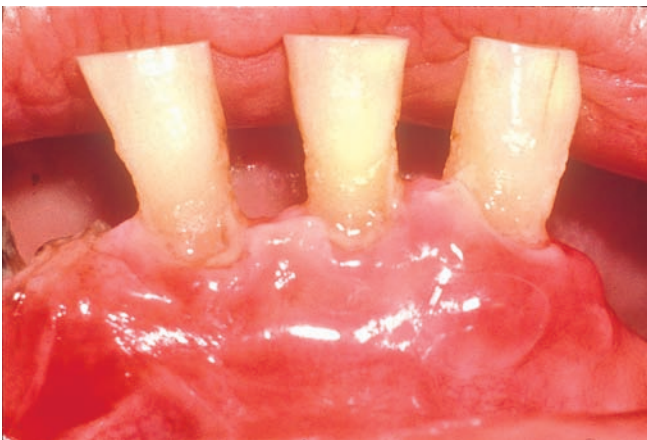


Fig. 76.5. Cicatricial pemphigoid: intact bulla.



Fig. 76.6. Cicatricial pemphigoid: sloughing gingiva.



Fig. 76.7. Cicatricial pemphigoid: positive Nikolsky sign.



Fig. 76.8. Symblepharon: a lid-eye adhesion in pemphigoid.

ULCERATIVE LESIONS

Traumatic Ulcer (Figs. 77.1–77.3) Recurrent oral ulceration is a common condition caused by several factors, primarily trauma. Ulcers may occur at any age and in either sex. Likely locations for traumatic ulcers are the labial/buccal mucosa, palate, and borders of the tongue.

Traumatic ulcers may result from chemicals, heat, or mechanical force and are often classified according to the exact nature of the insult. Pressure from an ill-fitting denture base or flange or from a partial denture framework is a source of a **decubitus** or **pressure ulcer**. **Trophic**—or **ischemic**—**ulcers** occur particularly on the palate at the site of a previous injection. Dental injections have also been implicated in the traumatic ulcerations seen on the lower lip by children who chew their lip after dental appointments. In addition to factitial injury, young children and infants are prone to traumatic ulcers of the soft palate from thumb sucking, called **Bednar aphthae**.

Ulcers may be precipitated by contact with a fractured tooth or restoration, a partial denture clasp, or inadvertent biting of the mucosa. Palatal ulcers appear after the mucosa is burned by hot food or drink. Other traumatic ulcers are caused by factitial injury from inappropriate use of fingernails or other objects on the oral mucosa. The diagnosis is simple and often established from a careful history and examination of the physical findings.

The appearance of a mechanically induced traumatic ulcer varies according to the intensity and size of the agent. The ulcer usually appears slightly depressed and oval. An erythematous zone is initially found at the periphery; this zone progressively lightens as the ulcer heals. The center of the ulcer is usually yellow-gray. Chemically damaged mucosa, such as that seen with an aspirin burn, is less well defined and contains a loosely adherent, coagulated, white surface slough. After removal of the traumatic influence, the ulcer should heal within 2 weeks. If healing does not occur, other causes should be suspected and a biopsy should be performed.

Recurrent Aphthous Stomatitis (Minor Aphthae, Aphthous Ulcer) (Figs. 77.4–77.6) **Recurrent aphthous stomatitis** is characterized by recurrent, painful ulcers of the oral mucosa. The disorder is classified into three categories according to size: **minor aphthae**, **major aphthae**, and **herpetiform ulcers**. Approximately 20% of the population is afflicted with minor aphthae, or *canker sores*, as they are commonly called. They may be seen in anyone, but women and young adults are slightly more susceptible. Familial patterns have been demonstrated, and persons who smoke are less frequently affected than nonsmokers. Although the cause is unknown, studies suggest an immunologic process involving T-cell-mediated cytolytic activity and tumor necrosis factor in response to human leukocyte antigen or foreign antigens. Factors that promote thinned mucosa (trauma, endocrinopathies, menstruation, nutritional deficiencies), immune dysfunction (atopy, stress), or exposure to antigens (food allergies)

contribute to the presentation of antigens to Langerhans cells and the abnormal T-cell response.

Minor aphthae have a propensity for movable mucosa that is situated over minor salivary gland tissue. The labial, buccal, and vestibular mucosa are frequently affected, as are the fauces, tongue, and soft palate. Ulcers are rarely seen on keratinized mucosa, such as the gingiva and hard palate. Prodromal symptoms of paresthesia or hyperesthesia are sometimes reported. The ulcers are shallow, yellow-gray, oval, well demarcated, and small (<1 cm; usually about 3–5 mm in diameter). A prominent erythematous border surrounds the fibrinous pseudomembrane. No vesicle formation is seen in this disease—a distinctive diagnostic feature. Ulcers that occur along the mucobuccal fold often appear more elongated. Burning is a preliminary symptom that is followed by intense pain lasting a few days. Tender submandibular, anterior cervical, and parotid lymph nodes are sometimes present, particularly when the ulcer becomes secondarily infected.

Aphthae are recurrent, and the pattern of occurrence varies. Most persons exhibit single ulcers once or twice a year, beginning during childhood or adolescence. The ulcers occasionally appear in crops, but usually fewer than five occur at one time.

Minor aphthae usually heal spontaneously without scar formation within 14 days. Some patients have multiple ulcers during a period of several months. In these cases, ulcers are in various stages of erupting and healing and produce constant pain. Although no medication has been totally successful for treating aphthous stomatitis, patients have responded to 5% amlexanox (Aphthasol), topical corticosteroids, and coagulating and cauterizing agents, as well as specific food avoidance.

Pseudoaphthous (Figs. 77.7 and 77.8) **Pseudoaphthae**, a term coined by Binney, refers to recurrent, aphthouslike mucosal ulcers of the mouth that are associated with nutritional deficiency states. Studies indicate that 20% of patients with recurrent aphthous stomatitis are deficient in folic acid, iron, or vitamin B₁₂. Pseudoaphthae are frequently seen with inflammatory bowel disease, Crohn disease, gluten intolerance (celiac disease), and pernicious anemia.

Pseudoaphthae resemble both minor and major aphthous stomatitis but are characteristically more persistent. There is a slight predilection for women between 25 and 50 years of age. The ulcers are depressed, rounded, and painful and are sometimes multiple in number. The borders may be raised, firm, and irregular. Occasionally lesions are accompanied by mucosal fissures and nodules. Alterations of the tongue papillae may suggest an underlying nutritional deficiency state. Healing is slow, and patients may report that they are rarely free of ulceration. Chronic and persistent presence of aphthae necessitates evaluation for nutritional deficiencies, including hematologic studies. If the laboratory results are abnormal, a medical referral is required.



Fig. 77.1. Traumatic ulcer: denture flange-induced.*



Fig. 77.2. Traumatic ulcer: molar area, under denture.*



Fig. 77.3. Traumatic ulcer: irregular, caused by hot food.

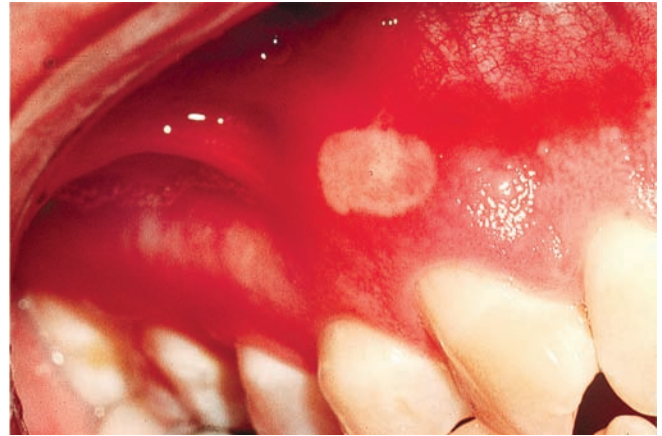


Fig. 77.4. Aphthous: oval ulcer on alveolar mucosa.



Fig. 77.5. Aphthous: prominent red border on labial mucosa.

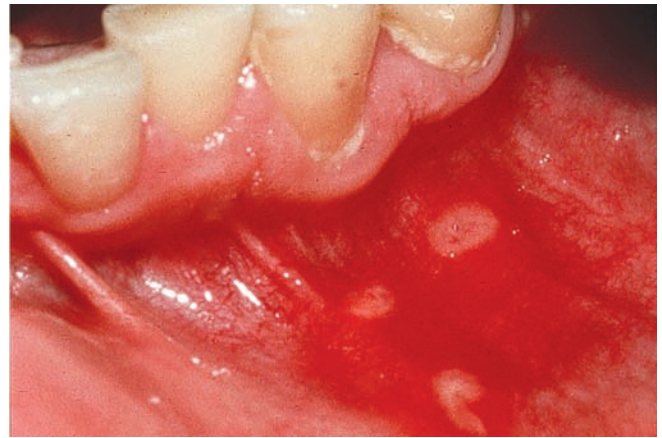


Fig. 77.6. Aphthae: a cluster of ulcers with typical shapes.



Fig. 77.7. Pseudoaphthous: irregular ulcer in Crohn disease.



Fig. 77.8. Pseudoaphthae: corrugated ulcers, Crohn disease.

ULCERATIVE LESIONS

Major Aphthous (Figs. 78.1–78.4) **Major aphthous** is a severe form of aphthous stomatitis that produces larger (≥ 1 cm), more destructive, and deeper ulcers that last longer and recur more frequently than minor aphthae. The cause is the same as for minor aphthae, namely, an immune defect in T-lymphocyte function. Young women with anxious personality traits and HIV-seropositive persons are commonly affected. Historically, this condition was also known as **Sutton disease** or **peradenitis mucosa necrotica recurrens** (PMNR).

Major aphthae are usually multiple. They involve the soft palate, tonsillar fauces, labial mucosa, and tongue and occasionally extend onto the attached gingiva. Characteristically, the ulcers are crateriform, asymmetric, and unilateral. The most prominent feature is the large size together with a depressed, necrotic center. A red raised inflammatory border is common. Depending on size, traumatic influences, and secondary infection, ulcers may last from several weeks to months. Because the ulcers erode deep into the connective tissue, they may heal with scar formation after repeated recurrences. Muscle destruction can result in tissue fenestration. If the periodontium is involved, tissue attachment may be damaged. Extreme pain and lymphadenopathy are common symptoms.

Use of steroids (topical, intralesional, or systemic) can accelerate healing and reduce scarring. Ulcers similar to those of major aphthae are seen with some frequency in association with cyclic neutropenia, agranulocytosis, and gluten intolerance. Ulcers located on the tongue may strongly resemble carcinoma. The presence of scarring is of diagnostic importance to rule out a malignant condition.

Herpetiform Ulceration (Figs. 78.5 and 78.6) **Herpetiform ulceration** is the least common variant of aphthous stomatitis. Clinically, the ulcers resemble those seen in primary herpes (hence the name *herpetiform*). The most prominent feature of the disease is the numerous, pin-head-sized, gray-white erosions that enlarge and coalesce into ulcers. Initially, the ulcers are 1 to 2 mm in diameter and occur in clusters of 10 to 100. The mucosa adjacent to the ulcer is erythematous, and pain is a predictable symptom.

Any part of the oral mucosa may be affected by herpetiform ulcerations, but the tip and margins of the tongue, and labial mucosa are particularly affected. The smaller size of these ulcerations distinguishes them from minor aphthae, and the absence of vesicles and gingivitis together with their frequent and recurrent nature distinguishes them from primary herpes and other oral viral infections. Virus cannot be cultured from these lesions, and the ulcers are not contagious.

The first episode of herpetiform ulceration usually occurs in patients in their late twenties and thirties, 10 years after the peak incidence of minor aphthae. The duration of recurrent attacks is variable and unpre-

dictable. Most patients experience healing within 2 weeks; however, some patients have constant lesions for months. The trigger of this disease has yet to be determined. Recurrent herpetiform ulcerations respond especially well to tetracycline suspensions, both topically and systemically, and the condition often regresses spontaneously after several years.

Behçet Syndrome (Oculo-Oral-Genital Syndrome) (Figs. 78.7 and 78.8) **Behçet syndrome**, named for the Turkish physician who described the ulcerative disorder, principally involves the oral cavity, eye, and genitals. Although the condition primarily presents affecting these three sites, it is now considered a multisystem disorder. In its fully developed state, cutaneous rashes, arthritis of the major joints, gastrointestinal ulcerations, cardiovascular disease, thrombophlebitis, and neurologic manifestations (headaches) are seen, although rarely are all components present in the same patient. The syndrome appears to be the result of a delayed hypersensitivity reaction, immune complexes, and vasculitis triggered by the presentation of human leukocyte antigens (HLA-B51) or environmental antigens, such as viruses, bacteria, chemicals, heavy metals, or pesticides. Behçet syndrome is two times more prevalent in men than in women and typically develops between 20 and 30 years of age. Persons from Asia, Eastern Mediterranean countries (along the ancient Silk Route), and Great Britain are most commonly affected.

Eye manifestations of Behçet syndrome include photophobia, conjunctivitis, and posterior uveitis. Rarely, pus in the anterior eye chamber (hypopyon) occurs that can lead to blindness. Eye lesions may be concurrent or occur years after oral and genital ulcers. Skin changes are characterized by subcutaneous nodules and macular and papular eruptions that vesiculate, ulcerate, and encrustate. Genital ulcers may involve the mucosa or skin and tend to be smaller and less common than the oral lesions.

Oral ulcers, the most prevalent lesion of Behçet syndrome, are the initial sign of disease in about 50% of patients. Although they may be any of the three forms of aphthous stomatitis, they most often occur in crops of six or more on the soft palate and oropharynx (infrequent sites for routine aphthae). Characteristically, the ulcers are recurrent, shallow, oval, and variable in size. Small lesions tend to occur more frequently than larger lesions. A serofibrinous exudate covers the surface, and the margins are red and well demarcated. Patients frequently report pain, and recurrent periods of exacerbation and remissions are characteristic. Topical and systemic steroids are used to treat the symptoms of patients with limited mucocutaneous involvement. Protracted disease involving the neuro-ocular structures requires the care of a physician. Azathioprine, cyclophosphamide, thalidomide, and colchicine have been used successfully in select cases. All of these agents have potentially serious side effects; they are best prescribed by experts.



Fig. 78.1. Major aphthous: persistent ulcers on gingiva.*



Fig. 78.2. Major aphthous: multiple, irregular tongue ulcers.*



Fig. 78.3. Major aphthous: deep, painful gingival ulcers.*

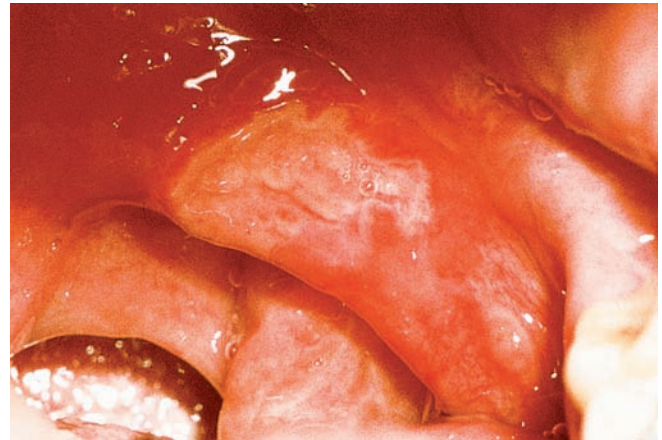


Fig. 78.4. Major aphthous: large irregular ulcer, soft palate.*



Fig. 78.5. Herpetiform ulceration: many ulcers of mucosa.‡



Fig. 78.6. Herpetiform ulceration: small crops, labial mucosa.‡

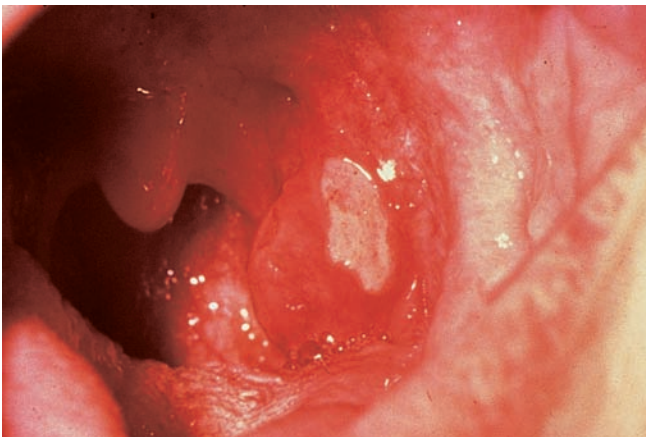


Fig. 78.7. Behçet syndrome: 27-year-old man, tonsillar ulcer.



Fig. 78.8. Behçet syndrome: genital ulcers.

ULCERATIVE LESIONS

Granulomatous Ulcer (Figs. 79.1 and 79.2) Granulomatous infections may produce oral ulcers. Two of the more common oral granulomatous infections are **tuberculosis** and **histoplasmosis**. These two infections exhibit primary, latent, and reactivated states. Although the oral lesions are relatively uncommon, they occur with frequency when infected respiratory secretions are implanted into oral mucosa following trauma. Adults with advanced pulmonary disease and persons with acquired immune deficiency syndrome (AIDS) are more often affected. Pulmonary lesions usually precede oral lesions. Thus, symptoms of persistent cough, fever, night sweats, weight loss, and chest pain are important historic findings.

Dissemination of organisms (*Mycobacterium tuberculosis* or *Histoplasma capsulatum*) from the lungs via infected sputum is the primary mode of oral infection. The classic oral manifestation of tuberculosis and histoplasmosis infection is a chronic nonhealing ulcer. Most oral tuberculous ulcers occur on the dorsum of the tongue, labial mucosa at the commissure, gingiva, and palate. Oral ulcers of histoplasmosis most often occur on the tongue, palate, and buccal mucosa. The clinical picture varies. The ulcer may resemble a traumatic ulcer or epidermoid carcinoma, particularly when the location is on the lateral tongue border. Lesions on the alveolar ridge often resemble a granulating extraction site. The center of the ulcer is necrotic, yellow-gray, and depressed. The periphery of the ulcer is undulating or lumpy and cobblestoned. The margin of the lesion is irregular, well demarcated, and undermined. Nodular and vegetative components are often seen in conjunction with the ulcers of histoplasmosis. Cervical lymphadenopathy is a common finding. Some patients report pain; in these patients, the discovery may be an incidental finding. Other patients experience severe, unremitting discomfort or bony involvement. Tuberculous and histoplasmosis lesions are contagious; the organisms can be transmitted by coughing and aerosols.

A biopsy or culturing is required to confirm the diagnosis. Histologic features and special stains (Ziehl-Neelsen for tuberculosis) demonstrate the presence of the causative organisms and groups of macrophages organized into granulomas. Treatment of the primary lung problem is with specific long-term antibiotics. For tuberculosis, isoniazid, rifampin, rifapentine, ethambutol, streptomycin, and pyrazinamide are administered. Drug combinations are selected based on drug resistance exhibited by the infecting organism. For histoplasmosis, amphotericin B, ketoconazole, or fluconazole are administered. The lung disease should be effectively treated before initiating dental treatment.

Squamous Cell Carcinoma (Figs. 79.3–79.6) **Squamous cell carcinoma** often appears as a chronic, nonhealing ulcer. In early stages, the lesion is usually small, nonpainful, and nonulcerative. However, the persistent nature of the disease results in neoplastic proliferation

that soon exhausts the blood supply, resulting in surface telangiectasia and eventual ulcer formation. Older ulcers tend to be large, crateriform, granular, and covered by a central yellow-gray necrotic slough. Red, raw foci are frequent. The borders are firm, raised, irregular, and sometimes fungating or rolled.

Carcinomas may occur anywhere in the mouth. The most common sites are the posterior third of the lateral margin of the tongue and the floor of the mouth. The retromolar trigone, soft palate, and tonsillar fauces are also frequently affected. Associated features may include pain, numbness, leukoplakia, erythroplakia, induration, fixation, and regional lymphadenopathy. Metastatic lymphadenopathy is characterized by nonpainful rubbery or hard nodes that are fixed at the base and matted together. Excessive use of alcohol and tobacco heighten suspicion of oral carcinoma when a persistent ulcer does not heal within 14 days. Biopsy should be performed by the clinician, who provides the definitive treatment. Treatment involves surgery and radiation therapy.

Chemotherapeutic Ulcer (Figs. 79.7 and 79.8) Patients receiving immunosuppressant drugs for a variety of serious illnesses, including organ transplantation, autoimmune conditions, and neoplasia, may develop oral ulcerations and mucositis. Side effects of the chemotherapeutic drug may directly or indirectly harm the oral mucosa. Antimetabolites such as methotrexate inhibit the replication of rapidly reproducing cells, including the oral epithelium, whereas alkaloids such as cyclophosphamide induce leukopenia and secondary ulcer formation.

The chemotherapeutic ulcer, an early sign of drug toxicity, appears during the second week of therapy and usually persists for 2 weeks. These ulcers may occur on any oral mucosal site, but more commonly affect nonkeratinized mucosa (lips, buccal mucosa, tongue, floor of the mouth, and soft palate) before keratinized mucosa (gingival, hard palate, and dorsal tongue). The affected area initially turns red and burns. The surface epithelium is lost (mucositis), then a large, deep, necrotic, and painful ulcer forms. The margins of the ulcer are irregular, and the characteristic red inflammatory border is often not present because of the lack of an inflammatory response by the host. If the pain becomes severe and the intake of adequate nutrition and fluids is impaired, a reduction in chemotherapy drug dose may be necessary.

Culturing is highly recommended for all oral lesions in patients on chemotherapy because of the propensity for infection with gram-negative organisms, fungi, or recurrent herpes simplex virus. Topical anesthetics and Benadryl mouth rinses are used to minimize symptoms, whereas oral hygiene measures, including antimicrobial agents such as chlorhexidine, are critical to prevent secondary infection, soft tissue necrosis, and osseous necrosis. Consultation and open communication between the physician and the dentist can help reduce complications and promote oral comfort.



Fig. 79.1. Granulomatous ulcer: caused by *M. tuberculosis*.

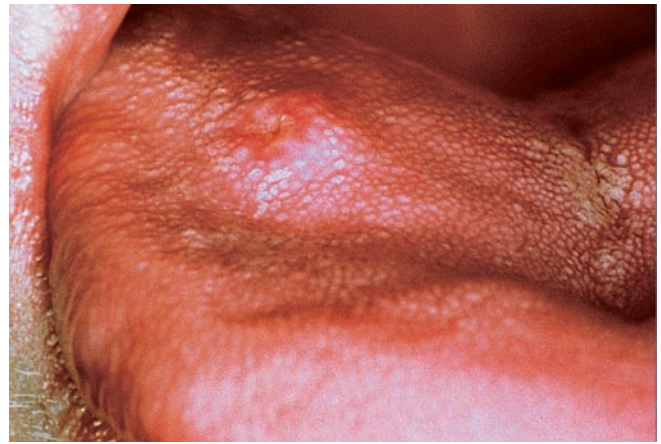


Fig. 79.2. Granulomatous ulcer: caused by histoplasmosis.



Fig. 79.3. Squamous cell carcinoma: indurated and raised.



Fig. 79.4. Squamous cell carcinoma: ulcer floor of mouth.



Fig. 79.5. Squamous cell carcinoma: at labial commissure.*

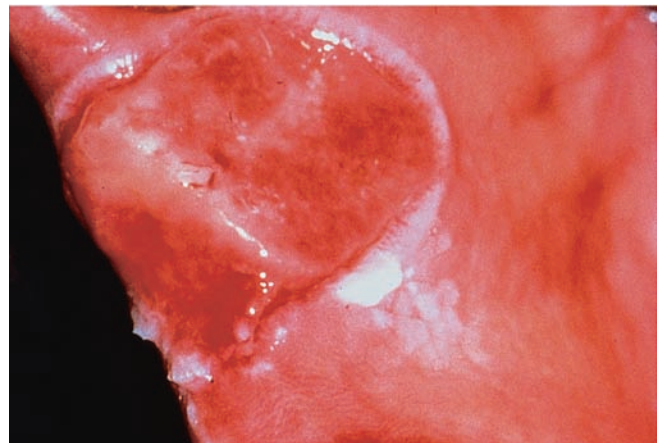


Fig. 79.6. Squamous cell carcinoma: spreading, buccal mucosa.*



Fig. 79.7. Chemotherapy-induced ulcer: in leukemia.

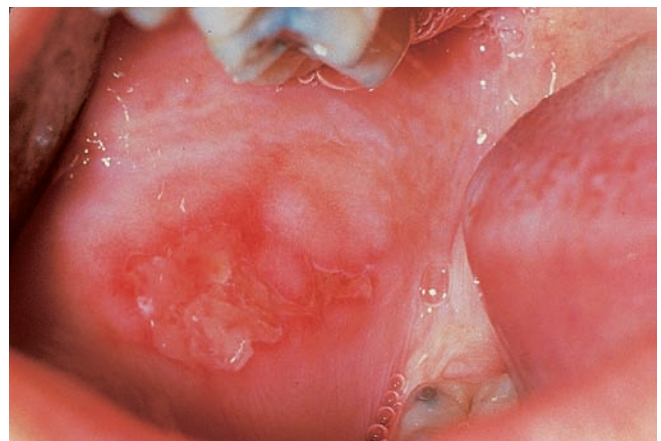


Fig. 79.8. Chemotherapy-induced ulcer: by methotrexate.

CASE STUDIES

CASE 19. (Fig. 79.9) This 67-year-old Caucasian woman presents to your office for the first time. She is edentulous and wants new dentures. Her current dentures are about 11 years old, and she has not been to a dentist in a while. She does not smoke tobacco, although she did for about 35 years. She quit 4 years ago. She drinks a glass of wine once a week. She is widowed and has dated a couple of men during the last 4 years, whom she has met at evening Moose lodge dances. She is asymptomatic and feels fine.



1. Describe the features in the clinical photograph.
2. Which term best describes the lesion?
 - A. Leukoplakia
 - B. Erythroplakia
 - C. Speckled erythroplakia
 - D. Ulcer
3. Why is it important to pull the tongue out during the examination?
4. Is this lesion a normal finding, a variant of normal, or disease? What clinical features are suggestive that this condition is benign or malignant?
5. What is the most likely diagnosis of this condition?
6. Which of the following would be *not* be expected to occur with this condition?
 - A. Induration
 - B. Rapid appearance
 - C. Lymphadenopathy
 - D. History of smoking
7. What factors place this patient at risk for this condition being neoplasia?

CASE 20. (Fig. 79.10) This 57-year-old woman presents with pain near the back of her maxillary denture. The pain has been present for about 5 days and makes it difficult for her to swallow. She works at a local factory in the accounting department. She had a cold last week, but feels like she is recovered. No other significant medical findings were noted on her medical history form.



1. Describe the oral findings.
2. What is the significance of the location of the lesion?
3. What questions could you ask regarding the information provided that might provide a diagnostic clue of what this condition is?
4. What is the significance of the lesion's red border?
5. What is the most likely diagnosis of this condition?
6. What is the cause of this condition?
7. What other conditions should be considered in the differential diagnosis?

Oral Manifestations of Sexual Conditions and Systemic Drug Therapies

Dental Objectives:

- Define oral entities that appear as a result of sexual activity, sexually transmitted infections, or drug therapy.
- Recognize the causes and clinical features of these conditions.
- Use the diagnostic process to distinguish similar-appearing oral conditions that result from sexual activity, sexually transmitted infections, or drug therapy.
- Recommend appropriate treatments for oral conditions that result from sexual activity, sexually transmitted infections, or drug therapy.

Dental Hygiene Objectives:

- Define oral entities that appear as a result of sexual activity, sexually transmitted infections, or drug therapy.
- Recognize the clinical features of these conditions.
- In the clinical setting, document the characteristics of oral condition discussed in this section in terms of:
 - a. Anatomic location
 - b. Color
 - c. Border
 - d. Configuration
 - e. Type
- Identify conditions discussed in this section that (1) require the attention of the dentist and/or (2) affect the delivery of periodontal debridement and oral hygiene measures.

SEXUALLY RELATED AND SEXUALLY TRANSMISSIBLE CONDITIONS

Traumatic Conditions (Figs. 80.1 and 80.2) Injury to the lingual frenum and soft palate are common oral conditions associated with sexual activity. Ulceration of the lingual frenum may occur when the tongue is rubbed against the incisal edge of the mandibular incisors during orogenital sexual activity. The ulcer has a gray-white fibrous coating and an erythematous border. A history of cunnilingus confirms the diagnosis, and abstinence is recommended to permit healing. Chronic irritation may lead to secondary bacterial infection, development of leukoplakia or a traumatic fibroma, or it may permit ingress of the human papillomavirus. Fellatio can traumatize oral soft tissues and produce erythema and submucosal hemorrhage of the soft palate. Isolated bright red petechiae initially appear. They eventually become a confluent patch (ecchymosis) that bridges the palatal midline. The purpuric patch is painless and does not blanch on diascopy. It clinically resembles the petechial patch produced by infectious mononucleosis. However, swollen lymph nodes and fever are characteristically absent. Petechiae darken and fade away in about a week.

Sexually Transmitted Pharyngitis (Figs. 80.3 and 80.4) **Pharyngitis** is inflammation of the back of the throat that is often due to infection. Venereal organisms, such as herpes simplex virus (HSV) type 2, *Neisseria gonorrhoeae*, and *Chlamydia trachomatis*, may cause pharyngitis by transmission from direct contact with infected genital or oral secretions or lesions. These infections occur most often in sexually active persons between 15 and 35 years of age. When primary HSV-2 infection manifests in the oral cavity, it produces inflammation of the pharynx and tonsils, fever, and generally less gingival inflammation than that caused by primary HSV-1 infection. Multiple small vesicles are usually apparent in the early stages; the vesicles collapse to form ulcers that resolve in 10 to 21 days. Antiviral agents during the first few days of clinical presentation are the treatment of choice.

N. gonorrhoeae infects nonkeratinized mucosal epithelium, producing a diffuse erythematous pharyngitis, small tonsillar pustules, or an erythematous and edematous patch involving the throat, tonsillar area, and uvula. Burning is the initial symptom, followed by increased salivary viscosity and halitosis. Other oral manifestations include painful, discrete ulcerations of the oral mucosa; fiery red and tender gingiva with or without necrosis of the interdental papilla; tongue ulcerations; and glossodynia. A single dose of ceftriaxone or cefixime or spectinomycin is effective in managing this condition. *C. trachomatis* may also cause a sore throat, mild pharyngitis, and tonsillar inflammation with pustule formation. Treatment is with azithromycin or doxycycline.

Infectious Mononucleosis (Figs. 80.5 and 80.6) **Infectious mononucleosis** is an acute viral infection characterized by fatigue, fever, sore throat, swollen lymph nodes, stomatitis, and occasional hepatosplenomegaly. It is

most commonly caused by the Epstein-Barr virus and occurs chiefly in adolescents and young adults. The disease is of low contagiousness, and transmission is through exchange of virus-contaminated saliva during deep kissing or sharing of straws. Oral lesions are often the earliest manifestations of infectious mononucleosis. Multiple red petechiae located at the junction of the hard and soft palate occur during the first few weeks of infection. These lesions turn brown and fade after several days. As the condition progresses, fatigue, exudative tonsillitis and bilateral, posterior, painful cervical lymphadenopathy become prominent findings. Less frequently, patients develop a rash, cough, necrotizing ulcerative gingivitis (NUG), or pharyngeal ulcers. Blood analysis reveals modest lymphocytosis, atypical lymphocytes, heterophile antibodies, and mildly elevated transaminase levels. Treatment is supportive and includes bed rest, soft diet, analgesics, and antipyretic agents. Recovery usually occurs within 1 to 2 months.

Syphilis (Figs. 80.7 and 80.8) **Syphilis** is a venereal disease caused by *Treponema pallidum*, an anaerobic spirochete. The hallmark of primary oral syphilis is the nonpainful **chancre**, which represents a granulomatous reaction to vascular obliteration. Chancres may affect any oral soft tissue. However, the lips are the most common site of involvement, followed by the tongue, palate, gingiva, and tonsils. Oral syphilis is more often observed in sexually active young men.

The **syphilitic chancre** initially appears as a small solitary papule that elevates, enlarges, erodes, and ulcerates. The lesion is usually punched-out, indurated, and 2 or 3 cm in diameter and lacks a red inflammatory border. The surface is covered by a yellowish, highly infectious serous discharge. Palatal erythema or an asymptomatic, reddish ulcer may be the initial lesion, along with swollen, nontender, firm, anterior cervical lymph nodes. Chancres typically persist for 2 to 4 weeks and heal spontaneously, causing patients to erroneously believe that treatment is not necessary. After a latent period of 4 weeks to 6 months, the **secondary stage of syphilis** appears. During this stage, the patient may report headaches, tearing of the eyes, nasal discharge, sore throat, generalized joint pain, enlarged lymph nodes, elevated temperature, and weight loss. A painless, symmetric maculopapular skin rash on both the palms of the hands and bottoms of the feet soon follows. Concurrent oral lesions of secondary syphilis appear as oval red macules, pharyngitis, or isolated or multiple mucous patches (painless, shallow, highly infectious ulcers surrounded by an erythematous halo). The borders are often irregular and resemble snail tracks. **Tertiary syphilis** occurs in infected persons many years after nontreatment of secondary syphilis. It is primarily characterized by palatal perforation and neurologic symptoms. Parenteral penicillin G remains the drug of choice for treating all stages of syphilis.



Fig. 80.1. Traumatic ulcer: of lingual frenum.



Fig. 80.2. Condyloma acuminatum: of lingual frenum.

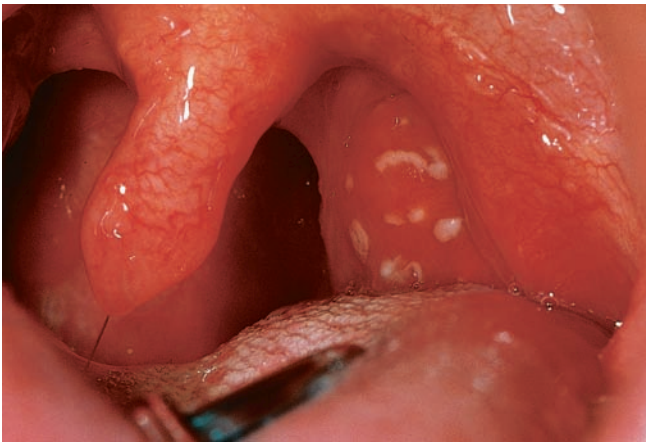


Fig. 80.3. Sexually transmitted HSV-2 pharyngitis.*

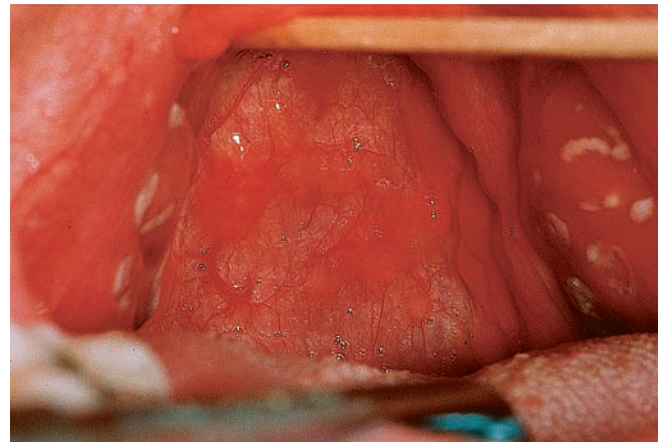


Fig. 80.4. Primary HSV-2 infection.*



Fig. 80.5. Infectious mononucleosis: palatal petechiae.



Fig. 80.6. Mononucleosis: exudative tonsillitis.



Fig. 80.7. Chancres of primary syphilis: split papule.

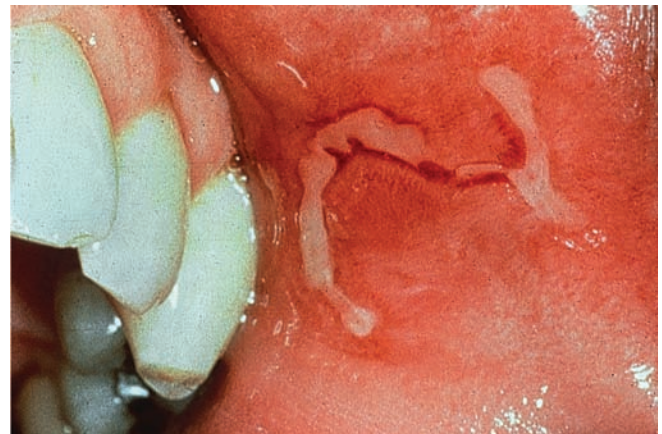


Fig. 80.8. Mucous patch secondary syphilis: snail track.

HIV INFECTION AND AIDS

Acquired Immunodeficiency Syndrome (AIDS) **AIDS** is a communicable disease caused by the human immunodeficiency virus (HIV), first reported by the Centers for Disease Control in 1981. HIV is an RNA virus that infects CD4⁺ T lymphocytes, brain glial cells, and macrophages. The virus is harbored in blood, tears, saliva, breast milk, and other bodily fluids and tissues of infected persons. It is predominantly spread by sexual contact, blood or blood products, or perinatally. Infection results by exposure to the virus through sharing of injected needles during drug use, having unprotected sex, receiving infected blood or blood products, or being accidentally exposed to infected materials.

In most cases, flulike symptoms develop 2 to 6 weeks after infection; then *persistent, generalized lymphadenopathy* occurs, followed by a latent phase. Initially, the latent phase is asymptomatic. Later lymphadenopathy, respiratory infections, weight loss, fever, chronic diarrhea, fatigue, skin anergy, oral candidiasis, hairy leukoplakia, parotid enlargement, and recurrent herpes virus infections develop. AIDS is defined as immune deficiency caused by HIV infection when CD4⁺ T-cell counts decrease to <200 cells/mm³ or when one of 30 opportunistic infections or certain forms of cancer develop. Treatment involves the use of antiretroviral therapy (ART), such as nucleoside analogs, protease inhibitors, fusion inhibitors, and integrase inhibitors in combination to block virus replication and maturation. These drugs have extended the lives of infected patients for more than 20 years. Oral manifestations of HIV infection are often numerous, concurrent, and recurrent. Recognition of the oral features warrants patient referral to a physician.

Oral Bacterial Infections (Figs. 81.1–81.3) Oral bacterial infections in HIV-infected patients often involve periodontal tissues. These include two necrotizing periodontal diseases discussed below.

Necrotizing Ulcerative Gingivitis (Figs. 81.1 and 81.2) **NUG** is common in HIV-infected patients. It is characterized by sudden onset of fiery red, swollen, painful, bleeding gingiva and a fetor oris. The interdental papillae are blunted or punched out, ulcerated, and covered by a grayish necrotic slough, without loss of bone. Fusiform and spirochetal organisms and immunosuppression are contributing causes. The affected gingiva responds quickly to debridement. Metronidazole is used when constitutional signs (fever, malaise, and lymphadenopathy) are present.

Necrotizing Ulcerative Periodontitis (NUP) (Fig. 81.3) **Necrotizing ulcerative periodontitis** is a rapidly progressive and destructive loss of periodontal attachment and bone. It initially manifests in the anterior periodontal tissues, then radiates to the posterior areas and has a distinct propensity for the incisor and molar teeth. This bacterial infection (caused by typical and atypical pathogens) is associated with pronounced immunosuppression. It

is characterized by pain and spontaneous gingival bleeding, gingival edema, ulceration and necrosis, rapid gingival recession (often interdentally), extremely rapid and irregular bone loss, delayed wound healing, and spread to adjacent mucosa. Aggressive periodontal measures and antibiotics are required for control.

HIV-infected patients are also prone to infections with bacterial flora uncommon to the oral cavity. The most commonly isolated bacteria are respiratory and coliform flora, *Klebsiella* species, and *Escherichia coli*. Infections by these organisms often produce diffuse, erythematous, and ulcerated changes of the tongue.

Oral Fungal Infections (Figs. 81.4–81.8) **Candidiasis** is a mild fungal infection of the skin or mucous membranes caused by an overgrowth of *C. albicans*. Candidiasis is the most common oral mucosal infection of patients with AIDS and is often the first oral manifestation. Candidal infections are usually chronic and may appear red, white, flat, raised, or nodular. Any oral mucosal surface may be infected, but the palate, tongue, and buccal mucosa are most frequent sites. The different types of candidiasis are discussed below and in Figures 65.1–65.8.

Linear Gingival Erythema (Fig. 81.4) Is a candidal infection in immunosuppressed persons characterized by a distinct red linear band along the marginal gingiva. It can also present on attached and nonattached gingiva as patches of tiny red or dark-red spots. Classically, the persistent linear red bands appear in the absence of apparent local factors such as plaque. The maxilla and mandible are equally affected. Spontaneous gingival bleeding and lack of response to conventional therapy are common unless antifungal agents are used in combination with plaque removal.

Pseudomembranous Candidiasis (Figs. 81.5–81.8) **Pseudomembranous candidiasis** is characterized by creamy-white plaques that on scraping reveal a red, raw, or bleeding mucosal surface. The organisms when smeared and stained with potassium hydroxide or cultured reveal hyphal forms typical of *Candida albicans*.

The **erythematous (atrophic)** form of candidiasis appears clinically as a diffuse red area, usually located on the dorsum of the tongue. At this location, it is associated with the loss of filiform papillae and is called **median rhomboid glossitis** (Fig. 81.7). A diffuse, erythematous contact lesion corresponding in size and shape to the tongue lesion may be apparent on the palate (Fig. 81.8). Although usually asymptomatic, patients may report mild discomfort, burning, or altered taste. **Chronic hyperplastic candidiasis**, a late stage of candidal infection, appears as nonpainful, diffuse, white keratotic plaques on the buccal mucosa (Fig. 65.4). The plaques cannot be wiped off. HIV-infected patients require systemic therapy with antifungal drugs. The disease is often chronic and recurrent and may predict esophageal candidiasis.



Fig. 811. HIV-associated necrotizing ulcerative gingivitis (NUG).



Fig. 812. HIV-associated necrotizing ulcerative gingivitis (NUG).

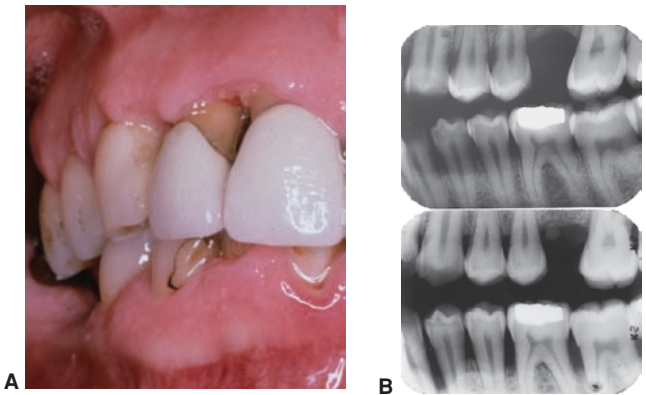


Fig. 813. NUP: A: anterior and (B) posterior bone loss in 6 months.



Fig. 814. Linear gingival erythema: on marginal and attached gingiva.



Fig. 815. Pseudomembranous candidiasis: in AIDS.[†]

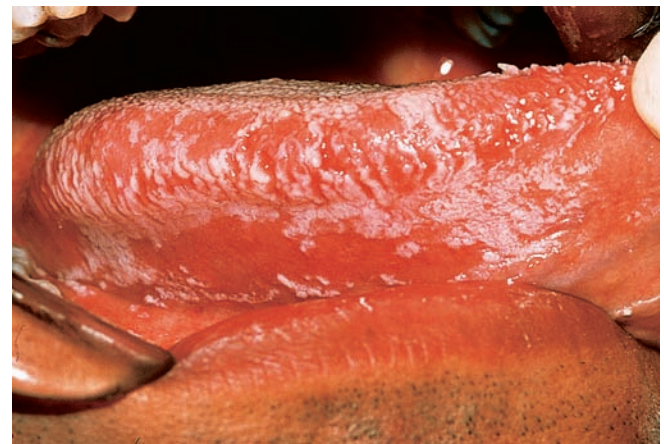


Fig. 816. Pseudomembranous candidiasis: in AIDS.[†]



Fig. 817. Median rhomboid glossitis (MRG): devoid of papillae.*



Fig. 818. Atrophic candidiasis: palatal lesion contacting the MRG.*

HIV INFECTION AND AIDS

Oral Viral Infections Human herpes viruses (HHVs), including HSV-1 and HSV-2, varicella-zoster, cytomegalovirus, Epstein-Barr virus, HHV-6, HHV-7, and HHV-8 figure prominently in acute and chronic oral disease in AIDS. **HSV infections (Figs. 82.1 and 82.2)** usually appear on the lips or in the mouth on keratinized epithelium. Recurrent infection forms small vesicles that rapidly erupt, leaving shallow yellow ulcers bordered by a red halo. Coalescence of adjacent vesicles into large ulcers is common. Unlike patients with normal immune function, patients with AIDS may have herpetic infections on mucosal surfaces typical of aphthous such as the tongue and buccal mucosa. Recurrent infections are more frequent, more persistent, and more severe (larger) in immunosuppressed patients.

Varicella-Zoster Virus (VZV; Fig. 82.3) **Varicella-zoster virus (VZV)** is a herpes virus that causes chickenpox and—in immunosuppressed adults—may reactivate to cause herpes zoster (shingles). VZV recrudesces more frequently in HIV-positive patients than the ordinary population. The clinical appearance is similar in both groups, but the prognosis is worse for immunosuppressed patients. VZV produces multiple vesicles that are commonly located on the trunk or face and are usually self-limiting and unilateral. Vesicles are typically found along a branch of the trigeminal nerve, inside or outside the mouth. Vesicle eruption, coalescence, pustule and ulcer formation, and scabbing are characteristic of the condition. Deep, searing pain is the premiere symptom and may persist after the lesions heal (**postherpetic neuralgia**). Antiviral agents are used to accelerate healing and alleviate symptoms.

Cytomegalovirus (CMV; Fig. 82.4) CMV infection occurs in nearly 100% of HIV-positive men who have sex with men and approximately 10% of children with AIDS. The virus has a predilection for secretory tissue (salivary glands) and is prevalent in the saliva of HIV-seropositive persons. Inflammatory changes associated with CMV in HIV-infected persons include unilateral and bilateral parotid gland swelling and xerostomia. CMV-induced oral ulcerations are nonspecific, often resemble aphthal, and can occur on any mucosal surface. They are more frequent when the CD4 count is <100 cells/mm³.

Epstein-Barr Virus (EBV) and Hairy Leukoplakia (Figs. 82.5, 45.7, and 45.8) Hairy leukoplakia is a raised, corrugated white lesion on the lateral border of the tongue that is associated with EBV and immunosuppression. Early lesions appear as discrete, white, vertically oriented plaques on the lateral borders of both sides of the tongue. Mature lesions may cover the entire lateral and dorsal surface of the tongue and extend onto the buccal mucosa and palate. The lesions are asymptomatic, cannot be rubbed off, and may pose an aesthetic problem to the patient. Histologic features are hyperkeratotic hairlike projections, koilocytosis (swollen epithelial cells), minimal inflammation, and, frequently, candidal coinfection. Electron microscopic examination shows EBV particles. Treatment is with antiviral agents.

Human Papillomavirus (HPV) Oral manifestations of HPV occur frequently in persons infected with HIV, even in spite of ART. So far, more than 100 serotypes of HPV have been identified. A variety of benign mucocutaneous lesions are induced by the virus, including squamous papilloma, verruca vulgaris, focal epithelial hyperplasia (Heck disease), and condyloma acuminatum. These entities are discussed below and in Section 10.

Condyloma Acuminatum (Fig. 82.6) The condyloma acuminatum, or venereal wart, is a slow-growing HPV-induced benign growth. It appears as a small, soft, pink to dirty-gray papule that has a cauliflowerlike surface. Lesions are often multiple, recurrent, and coalescent to form large, sessile, pebbly growths. They can be found on any mucosal surface, particularly the ventral tongue, gingiva, labial mucosa, and palate. Transmission is by (1) direct contact that results in contagious spread from anal or genital sites or (2) self-inoculation. Treatment consists of local excision or antiviral therapy together with simultaneous eradication of all lesions of infected partners.

Oral Malignancies (Figs. 82.7 and 82.8) **Kaposi sarcoma (Fig. 82.7)** is a tumor of vascular (endothelial) proliferation that affects the skin and mucosa. HHV-8, a herpesvirus capable of promoting angiogenesis, is the causative agent. Three types are recognized: indolent (classic), endemic (African), and immunosuppression associated. This tumor is the most common cancer associated with HIV infection, with at least 20% of all AIDS patients affected.

Kaposi sarcoma is characterized by three clinical stages. Initially, it appears as an asymptomatic red macule or patch. The tumor then enlarges into a red-blue plaque. Advanced lesions appear as lobulated, violet nodules that ulcerate and cause pain. Oral lesions are limited to the immunosuppression-associated form. The hard palate is the most common location, followed by the gingiva and buccal mucosa. Lesions are frequently multiple, uncomfortable, and aesthetically displeasing. Similar-appearing lesions, such as erythroplakia, purpura, hemangiomas, pyogenic granuloma, and bacillary angiomatosis, should be ruled out by biopsy. Localized radiation therapy and direct injection of chemotherapeutic drugs (vinblastine) or sclerosing agents have proven beneficial.

Non-Hodgkin Lymphoma (NHL, Fig. 82.8) and Squamous Cell Carcinoma **NHL** and **squamous cell carcinoma** are associated with HIV infection, probably as a result of abnormal immune surveillance and dysregulation of apoptosis. NHL is linked with EBV infection and often appears as a rapidly proliferating, purplish, nodular mass of the palatal-retromolar complex. Squamous cell carcinoma is most frequently found as a reddish white or ulcerated lesion on the posterolateral border of the tongue, floor of mouth (Fig. 79.4), or, less frequently, the gingiva. These neoplasms occur at a younger age when associated with HIV infection and when the usual cofactors—such as alcohol abuse, advanced age, and poor oral hygiene—are absent. Treatment involves chemotherapy and radiation.



Fig. 82.1. Recurrent herpes labialis: in AIDS.



Fig. 82.2. HIV-associated recurrent herpes simplex: ulcer.

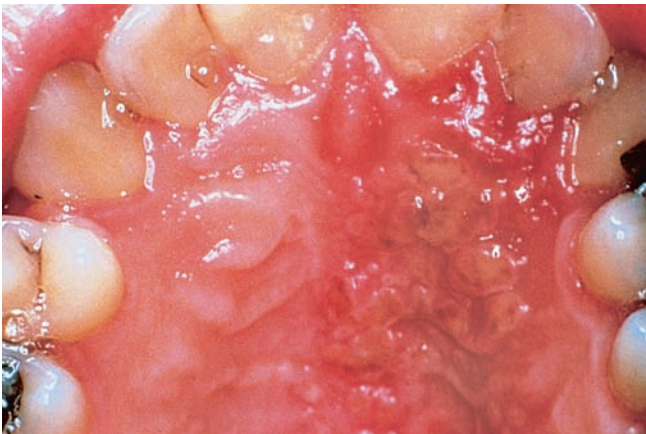


Fig. 82.3. HIV-associated herpes zoster: up to midline.



Fig. 82.4. Cytomegalovirus gingival ulceration.



Fig. 82.5. HIV-associated hairy leukoplakia: lateral tongue.



Fig. 82.6. HIV-associated condyloma acuminata.

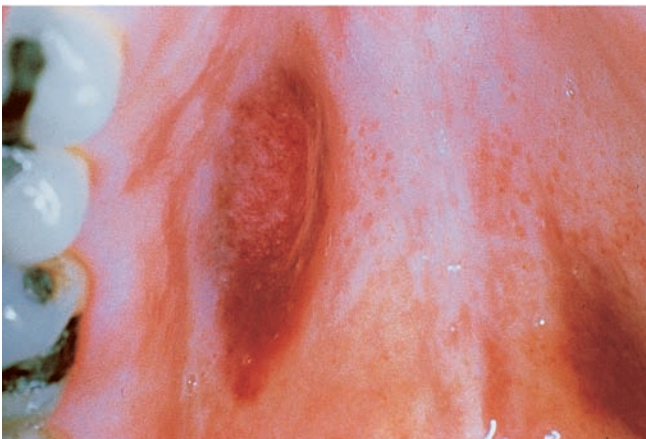


Fig. 82.7. HIV-associated Kaposi sarcoma: purple macules.

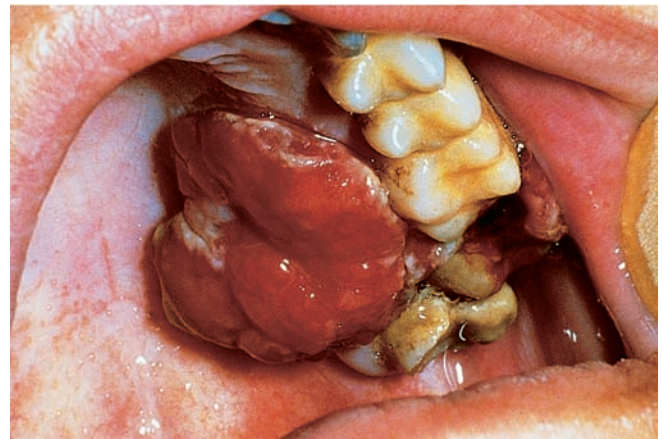


Fig. 82.8. HIV-associated non-Hodgkin lymphoma.

ORAL EFFECTS OF DRUGS AND THERAPIES

Meth Mouth (Figs. 83.1 and 83.2) Methamphetamine is a highly addictive substance that affects the central nervous system, causing euphoria and stimulation. It is a Schedule II narcotic that has a high potential for abuse. Common street forms of the drug are powder and crystal. The crystal form (also known as “ice,” “crystal,” and “glass”) has higher purity and more potential for abuse. Crystal is typically smoked, producing an immediate and intense sensation. The drug is also taken orally, by snorting or injected. The effects are rapid and generally last 4 to 12 hours. About 3% of young adults (ages 18 to 26) report use of crystal methamphetamine. Young adult users are disproportionately white or Native American and male. Methamphetamine use is associated with risky and antisocial behaviors, including other illicit drug use; criminal behavior; and several adverse health consequences, such as mood disturbances, insomnia, cardiovascular problems (hypertension), convulsions, weight loss, psychotic symptoms that can be long term, and risk for Parkinson disease.

Chronic “meth” use can have devastating effects on the mouth. It is associated with rampant caries (**meth mouth**), which results from the drug’s ability to decrease salivary flow or create the perception of a dry mouth and cause cravings for high-calorie carbonated soft drinks. Chronic exposure of the teeth to carbonated drinks produces a distinctive caries pattern often seen on the gingival and buccal surface of the teeth, especially the anterior teeth. Key signs include unaccounted and accelerated decay in teenagers and young adults, the distinctive decay pattern, and malnourished individuals. Clinicians should counsel patients to get professional assistance for substance abuse and also be aware that these patients (typical of drug abusers) may have a higher tolerance for local and general anesthetics.

Graft-Versus-Host Disease (Figs. 83.3 and 83.4) **Graft-versus-host disease (GVHD)** is a frequent and potentially serious complication of allogeneic bone marrow transplant. Allogeneic transplants involves transfer of tissue (in this case, hematopoietic stem cells) from different individuals of the same species. Donors are human leukocyte antigen (HLA) matched, as close as possible with the patient. The disease develops within the first 2 years of transplant as a result of cytotoxic T cells that target the graft as well as the skin, liver, lacrimal and salivary glands, and oral mucosa.

Oral GVHD is common and in some cases may be the only area affected. The presentations are varied and divided into acute (develops within the first 100 days) and chronic (after 100 days). Acute oral GVHD appears as mucosal erythema; atrophy; erosions; or severe sloughing of the tongue, buccal mucosa, or lips. Chronic oral GVHD often results in a lacy network of white lines or plaques that resemble lichen planus. A burning sensation may accompany the condition. Affected salivary glands exhibit reduced function resulting in dry mouth

and can result in small superficial mucocles inside the lip or on the palate. Attempts are made to prevent the disease with proper HLA matching and the use of immunosuppressive drugs. Treatment of existing disease requires therapies that reduce pain and inflammation as well as improve moisture and salivary flow.

Bisphosphonate Osteonecrosis (Figs. 83.5–83.7) **Bisphosphonate osteonecrosis** is a condition of exposed, necrotic (dead) bone in patients who take or previously took bisphosphonate drugs, in the absence of other causes. Bisphosphonates inhibit osteoclast function; thus, they inhibit bone resorption. They are used to stabilize osteoporosis. They are also used in the management of osteopenia, osteogenesis imperfecta, Paget disease, and cancers involving bone. These drugs are administered orally (alendronate, ibandronate, risedronate) or intravenously (pamidronate, zoledronate). The intravenous (IV) drugs help slow the destruction from multiple myeloma and bony metastases from breast and prostate cancer. The intravenous forms are more potent and associated with higher risk for osteonecrosis than the orally administered types. Although the exact cause of bisphosphonate osteonecrosis is not well defined, the most common trigger is oral trauma (dental extraction, implant placement, osseous surgery). Increased duration of drug use, smoking, alcohol use, corticosteroid therapy, diabetes, and poor oral hygiene may also increase risk of developing the disease.

The jaw lesions associated with bisphosphonates begin as delayed healing after an extraction or trauma. Clinically, an oral ulcer that contains an area of exposed, underlying dead bone is seen. Early-stage disease causes little or no discomfort; however, progression leads to pain in the majority of cases. Lesions are characterized by their persistent nature and poor response to treatments. Management involves implementation of (1) preventive measures (perform extractions and invasive procedures) before initiating drug therapy, (2) good oral hygiene measures and the performance of endodontic procedures in lieu of extractions during the course of therapy, and (3) oral antimicrobial rinses, pain medications, and drugs that control infection and bone necrosis in established cases.

Drug-Induced Hyperpigmentation (Fig. 83.8) Many drugs and conditions can cause hyperpigmentation of the mucosa (see **Pigmented Lesions, Figs. 66.1–68.8** for additional discussion). In this example, oral pigmentation is associated with administration of doxorubicin (Adriamycin). This chemotherapy drug is used in the treatment of many types of cancers. Hyperpigmentation of the skin and mucosa has also been reported in HIV-infected patients. These changes are due to increased melanin pigmentation associated with the medications taken, such as clofazimine, ketoconazole, pyrimethamine, and zidovudine (AZT). Chronic use of minocycline, estrogen, and anti-malarial drugs can also result in pigmented mucosa.



Fig. 83.1. Meth mouth: rampant decay incisors.



Fig. 83.2. Meth mouth: rampant and more advanced.

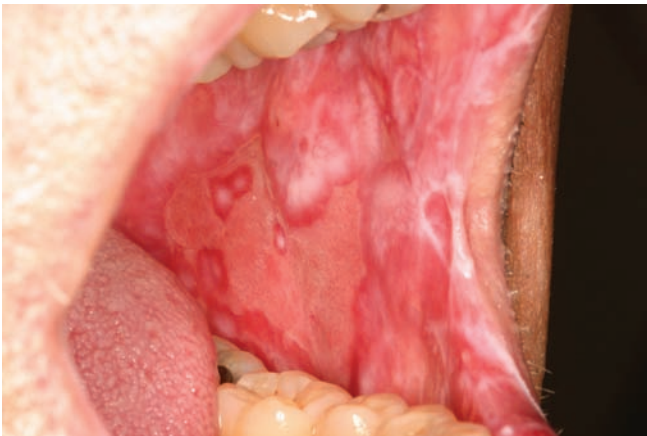


Fig. 83.3. Graft-versus-host disease: lichenoid mucosa.



Fig. 83.4. Graft-versus-host disease: mucocles on palate.



Fig. 83.5. Bisphosphonate osteonecrosis: exposed bone.



Fig. 83.6. Bisphosphonate osteonecrosis: dead bone molar region.



Fig. 83.7. Bisphosphonate osteonecrosis: draining onto face.



Fig. 83.8. Drug-induced hyperpigmentation: Adriamycin.

CASE STUDIES

CASE 21. (Fig. 83.9) This 27-year-old woman presents to your office as a new patient. She does not list her employment but has anterior crowns and with no visible decay. She claims her gums recently began hurting around several front teeth and now she can see space between some of her teeth. The condition is painful. She smokes a half a pack of cigarettes per day and claims to have been in drug rehabilitation once. She is divorced and has a 3-year-old son.

1. Describe the clinical state of the periodontium?
2. What clinical findings are suggestive of periodontal disease?
3. Which term best describes the lesion?
 - A. Necrotizing stomatitis
 - B. Necrotizing ulcerative gingivitis
 - C. Necrotizing ulcerative periodontitis
 - D. Rapidly progressive periodontitis
4. What factors (or condition) are associated with the oral findings?
5. Do you expect the condition to resolve if you clean her teeth?
6. If you refer this patient to a physician, what tests and drugs might they prescribe?



CASE 22. (Figs. 83.10) This 21-year-old college student presents for his 6-month cleaning. He is a senior at the local university and finishing his degree in business. He has a girlfriend who attends the same university. He has no significant medical findings and reports only a slightly sore throat.

1. Describe the oral findings.
2. How is the patient's medical history potentially relevant to the clinical condition?
3. What questions could you ask regarding the information provided that might provide a diagnostic clue of what this condition is?
4. The presence of this oral findings is suggestive of all of the following *except*:
 - A. Coughing
 - B. Fellatio
 - C. Bleeding disorder
 - D. Viral infection
 - E. Sturge-Weber syndrome
5. What is the most likely diagnosis of this condition?
6. What is the cause of this condition?
7. Why is knowledge of this sign important?
8. What complications can occur with this condition?



SECTION 12

Clinical Applications and Resources

Rx ABBREVIATIONS

<i>Abbreviation</i>	<i>English</i>	<i>Latin Derivative</i>
ad lib	at pleasure	ad libitum
a.c.	before meals	ante cibum
p.c.	after meals	post cibum
aq.	water	aqua
d.	a day, daily	dies
b.i.d.	twice a day	bis in die
t.i.d.	three times a day	ter in die
q.i.d.	four times a day	quater in die
h.	hour	hora
h.s.	at bedtime	hora somni
q.h.	every hour	quaque hora
q.3h.	every three hours	quaque tertia hora
q.4h.	every four hours	quaque quarta hora
q.6h.	every six hours	quaque sexta hora
n.r.	do not repeat	non repetatur
p.r.n.	as needed	pro re nata
stat.	immediately	statim
Sig.	label	signetur
c.	with	cum
gtt.	drops	guttae
tab	tablet	tabella
caps.	capsule	capsula
q.d.	every day	quaque die
p.o.	orally	per os

PRESCRIPTIONS AND THERAPEUTIC PROTOCOLS

Analgesics

<i>Generic Name and Concentration</i>	<i>Trade Name</i>	<i>Disp:</i>	<i>Sig:</i>
For Relief of Mild to Moderate Acute Pain			
Acetaminophen (Tylenol) 325 mg [OTC]	Tylenol	25	Take 2 tablets q.4h. p.r.n. for pain; not to exceed 12 tablets in 24 hours.
Aspirin 325 mg [OTC]		25	Take 2 tablets q.4h. p.r.n. for pain.
Ibuprofen (Motrin) 400 mg	Motrin	25	Take 2 tablets q.4h. p.r.n. for pain.
Propoxyphene napsylate 50 mg, and acetaminophen 325 mg	Darvocet-N 50	25	Take 2 tablets q.4h. p.r.n. for pain.
Naproxen sodium 220 mg [OTC]	Aleve	50	Take 2 tablets b.i.d p.r.n. for pain.
Naprosyn 375 mg			
Celecoxib 200 mg	Celebrex	18	Take 1 tablet q.d. for pain. Note: Do not prescribe if allergic to sulfa medications.
Diflunisal 500 mg	Dolobid	18	Take 1 tablet b.i.d. for pain.
For Relief of Moderate Acute Pain			
Acetaminophen 300 mg, with codeine 30 mg	Tylenol with Codeine No. 3	20	Take 1 to 2 tablets q.4h. p.r.n. for pain.
Aspirin 325 mg, with codeine 30 mg	Empirin with Codeine No. 3	20	Take 1 to 2 tablets q.4h. p.r.n. for pain.
Aspirin 325 mg, butalbital 50 mg, caffeine 40 mg	Fiorinal	20	Take 1 to 2 tablets q.4h. p.r.n. for pain.
Aspirin 325 mg, butalbital 50 mg, caffeine 40 mg, codeine 30 mg	Fiorinal with Codeine 30 mg	20	Take 1 to 2 tablets q.4h. p.r.n. for pain.
Acetaminophen 650 mg, with codeine 30 mg	Margesic No. 3	20	Take 1 to 2 tablets q.4h. p.r.n. for pain.
Hydrocodone bitartrate 5 mg, with acetaminophen 500 mg	Lortab 5, Anexsia 5/500, Vicodin, Zydone	20	Take 1 tablet q.4–6h. p.r.n. for pain.
Dihydrocodeine bitartrate 16 mg, aspirin 356.4 mg, caffeine 30 mg	Synalgos-DC Capsules	20	Take 1 to 2 tablets q.4h. p.r.n. for pain.
Hydrocodone 7.5 mg, ibuprofen 200 mg	Vicoprofen	20	Take 1 to 2 tablets q.4–6h. p.r.n. for pain.
Acetaminophen 325 mg, tramadol 37.5 mg	Ultracet	20	Take 2 tablets q.4–6 h. p.r.n. pain (not to exceed 8 tablets in 24 hr)
For Relief of Moderate to Severe Acute Pain			
Hydrocodone bitartrate 7.5 mg, with acetaminophen 500 mg	Lortab 7.5/500	30	Take 1 tablet q.6h. p.r.n. for pain.
Hydrocodone bitartrate 7.5 mg, with acetaminophen 650 mg	Lorcet Plus	30	Take 1 tablet q.6h. p.r.n. for pain.
Hydrocodone bitartrate 10 mg, with acetaminophen 650 mg	Lorcet 10/650	30	Take 1 tablet q.6h. p.r.n. for pain.
Oxycodone HCl 5 mg, with acetaminophen 325 mg	Percocet, Roxicet	24	Take 1 tablet q.6h. p.r.n. for pain.
Oxycodone HCl 5 mg, with acetaminophen 500 mg	Tylox	24	Take 1 tablet q.6h. p.r.n. for pain.
Oxycodone HCl 4.5 mg, oxycodone terephthalate 0.38 mg, aspirin 325 mg	Percodan	24	Take 1 tablet q.6h. p.r.n. for pain.
Meperidine HCl 50 mg, with promethazine HCl 25 mg	Mepergan Fortis Capsules	24	Take 1 tablet q.4–6h. p.r.n. for pain.

ANTIBIOTIC PROPHYLAXIS

To Prevent Infective Endocarditis—American Heart Association Guidelines Regimens for Dental Procedures (Single Dose 30 to 60 Minutes Before Procedure)

<i>Situation</i>	<i>Agent</i>	<i>Adults</i>	<i>Children</i>
Oral Dosage	Amoxicillin	2 g	50 mg/kg
Unable to take oral medication	Ampicillin or Cefazolin or ceftriaxone	2 g IM or IV 1 g IM or IV	50 mg/kg IM or IV 50 mg/kg IM or IV
Allergic to penicillins or ampicillin—oral	Cephalexin*† or Clindamycin or Azithromycin or Clarithromycin	2 g 600 mg 500 mg	50 mg/kg 20 mg/kg 15 mg/kg
Allergic to penicillins or ampicillin and unable to take oral medication	Cefazolin or Ceftriaxone† or Clindamycin	1 g IM or IV 600 mg IM or IV	50 mg/kg IM or IV 20 mg/kg IM or IV

* Or other first- or second-generation oral cephalosporin in equivalent adult or pediatric dosage.

† Cephalosporins should not be used in an individual with a history of immediate-type hypersensitivity to penicillin (i.e., anaphylaxis, angioedema of the airway).

Cardiac Conditions for Which Prophylaxis is Recommended during Dental Procedures

Prosthetic cardiac valve

Previous infective endocarditis

Congenital heart disease

- Unrepaired cyanotic congenital heart disease, including palliative shunts and conduits
- Completely repaired congenital heart disease defects with prosthetic material or device for first 6 months after procedure
- Repaired congenital heart disease with residual defects at the site or adjacent site of prosthetic patch/device (which inhibits endothelialization)
- Cardiac transplantation recipients who develop cardiac valvulopathy

Dental Procedures for which Infective Endocarditis Prophylaxis is Recommended

All dental procedures that involve "manipulation of gingival tissue or periapical region (root end) of teeth or perforation of the oral mucosa."

Dental Procedures for which Infective Endocarditis Prophylaxis is NOT Recommended

Routine anesthetic injection through noninfected tissue

Placement of removable appliances

Placement of orthodontic brackets

Bleeding from trauma to lips/ mucosa

Taking dental radiographs

Adjustment of orthodontic appliances

Shedding of deciduous teeth

ANTIBIOTIC THERAPY

To Eliminate Bacterial Organisms That Cause Oral Infection

Generic Name and Concentration	Trade Name	Disp:	Sig:
Phenoxymethyl penicillin 500 mg	Penicillin V	40	Take 2 tablets immediately and then 1 tablet q.6h. 1 hour a.c. Continue for 7 days.
Penicillin V potassium liquid 125 mg/5 mL	Penicillin VK	200 mL	Children should take 1 teaspoonful q.6h.
Amoxicillin 500 mg	Amoxil	40	Take 500-mg tablet t.i.d. Continue for 7 days.
Amoxicillin 500 mg, clavulanate potassium	Augmentin	30	Take 1 tablet 3 times daily for 7 days.
Cephalexin 500 mg	Keflex	56	Take 1 tablet q.6h. Continue for 7 days.
Dicloxacillin sodium 500 mg	Dynapen	40	Take 1 tablet q.6 h. Continue for 7 days.
<i>Note:</i> For penicillinase-resistant infection.			
Erythromycin ethylsuccinate 400 mg	E.E.S.	56	Take one 400-mg tablet q.6h. Continue for 7 days.
Trimethoprim 80 mg, with sulfamethoxazole 400 mg	Bactrim	40	Take 1 tablet q.12h. Continue for 7 days.
<i>Note:</i> For infections with <i>Escherichia coli</i> , <i>Haemophilus influenzae</i> , and <i>Klebsiella</i> and <i>Enterobacter</i> species.			
Metronidazole 500 mg	Flagyl	40	Take 2 tablets immediately, then 1 tablet q.6h. until gone.
<i>Note:</i> For febrile patients with acute necrotizing ulcerative gingivitis involving anaerobic bacteria.			
Clindamycin 300 mg	Cleocin	30	Take 1 capsule q.6–8 h. Continue for 7 days.
<i>Note:</i> For bone infections involving anaerobic bacteria.			
Tetracycline HCl V 250 mg	Achromycin	56	Take 1 tablet q.i.d. Continue for 7 days.
<i>Note:</i> For infections of periodontal tissues.			
*Use with caution in patients with diminished or impaired liver function and in patients with diminished or impaired kidney function			

ANTIMICROBIAL TOPICAL AGENTS AND RINSES

To Reduce Pathogenic Microbial Flora Often Associated with Periodontal Infections

<i>Generic Name and Concentration</i>	<i>Trade Name</i>	<i>Disp:</i>	<i>Sig:</i>
Chlorhexidine gluconate 0.12 % oral rinse	Peridex, PerioGard	480 mL	Swish 1 teaspoonful for 1 minute, then expectorate. Perform b.i.d. daily (morning and evening) after brushing teeth. Avoid eating or drinking for 30 minutes.
Tetracycline HCl 125 mg/ 5 mL	Sumycin	480 mL	Rinse with 2 teaspoonfuls for 3 minutes and swallow. Repeat q.i.d.
Chlorhexidine gluconate 2.5 mg	PerioChip	Box of 10 chips	(For in office use only.) Place in gingival sulcus in affected areas (≥ 5 mm).
Metronidazole 250 mg	Flagyl	28	Take 1 or 2 tablets q.i.d. for 7 to 10 days.
<i>Note:</i> Not recommended for children.			

ANTIFUNGAL THERAPY

To Eliminate Pathogenic Fungal Organisms and Re-establish Normal Oral Flora

Generic Name and Concentration	Trade Name	Disp:	Sig:
Nystatin vaginal tablets 100,000 IU	Nilstat	90	Dissolve 1 to 2 tablets as a lozenge in mouth 5 times d. for 14 consecutive days.
Nystatin 500,000 U	Mycostatin	60	Take 1 tablet by mouth daily for 14 consecutive days.
Clotrimazole 10 mg	Mycelex Troches	60	Dissolve 1 tablet as a lozenge 5 times d. for 14 consecutive days.
Ketoconazole* 200 mg	Nizoral	14	Take 1 tablet q.d. for 2 weeks.
Fluconazole** 100 mg	Diflucan	14	Take 2 tablets the first day, then 1 tablet q.d. for at least 2 weeks.
Itraconazole** 20 mg/mL	Sporanox Oral Solution	280 mL	Take 200 mg (20 mL)/day p.o. for 1 to 2 weeks. Vigorously swish solution in mouth for several seconds and swallow.
Nystatin topical powder 100,000 IU	Mycostatin	15 g squeeze bottle	Apply liberally to tissue side of clean denture p.c. Soak the clean denture in a suspension of 1 teaspoon of powder and 8 oz. of water overnight.
Nystatin ointment 100,000 IU	Mycostatin	15 or 30 g tube	Apply liberally to affected area 4–6 times d.
Ketoconazole cream 2%	Nizoral	15 g tube	Apply liberally to affected area after meals.
Betamethasone dipropionate 0.05% and clotrimazole 1%	Lotrisone	15 or 30 g tube	Apply liberally to affected area 4 or 5 times d.
Iodoquinol 10 mg, and hydrocortisone 10 mg ointment	Dermazene	15 or 30 g tube	Apply liberally to affected area 4 or 5 times d.
Nystatin 100,000 U and triamcinolone acetonide 0.1%	Mycolog II	15, 30 or 60 g tube	Apply liberally to affected area t.i.d. to q.i.d.

These drugs are most effective when dentures are removed and treated, if applicable, and when intake of fermentable carbohydrates is reduced.

*Use with caution in patients with diminished or impaired liver function. Also, beware of multiple drug interactions.

**Use with caution in patients with diminished or impaired liver function

ANTIVIRAL THERAPY

To Prevent Oral Herpetic Infections (Daily Suppression)

<i>Generic Name and Concentration</i>	<i>Trade Name</i>	<i>Disp:</i>	<i>Sig:</i>
Sunscreen SPF 30 or higher		1 bottle	Applied daily to sun exposed skin surface that are susceptible to recurrences
Acyclovir 200 mg	Zovirax	100	Take 1 tablet 5 times d.
Acyclovir 800 mg	Zovirax	100	Take 1 tablet b.i.d.
Valacyclovir 500 mg	Valtrex	100	Take 1 tablet q.d. or b.i.d.

To Prevent Oral Herpetic Infections Associated with Dental Procedures

Valacyclovir 500 mg	Valtrex	30	Take 4 capsules b.i.d. on day of dental treatment and 2 capsules b.i.d. the following day
---------------------	---------	----	---

To Treat Oral Herpetic Infections

Topical

Acyclovir cream 5%	Zovirax	15 g	Apply to oral lesions with a cotton-tip applicator 6 times d.
Penciclovir 1% ointment	Denavir	15 g	Apply to lesions with a cotton-tip applicator at least 5 times d.
Foscarnet* 3% cream	Foscavir	Compounded	Apply to lesions with a cotton-tip applicator at least 5 times d.
<i>Note: Used only if lesions are known to be acyclovir-resistant.</i>			
Docosanol 10% cream	Abreva	2 g	Apply to lesions with a cotton-tip applicator at least 5 times d.
Benzalkonium chloride	Viroxyn	1 tube	Remove cap, squeeze tube, allow tip to saturate swab, then apply to developing lesion.

Systemic

Acyclovir 200 mg, 400 mg, 800 mg	Zovirax	50	Take 800 mg at onset of recurrence and 800 mg b.i.d. for at least 3 days.
Famciclovir 500 mg	Famvir	50	Take 1 capsule at onset of recurrence and t.i.d. for at least 3 days
Valacyclovir 500 mg	Valtrex	50	Take 4 tablets at onset of recurrence and 2 to 4 tablets 12 hours later.
L-Lysine 500 mg	Enisyl	100	Take 4 tablets q.4h. until symptoms subside.

Note: Treatment should begin during the early stage of the recurrence. Most effective if foods containing high content of arginine are avoided.

To Treat Oral Human Papillomavirus Infection

Generic Name and Concentration	Trade Name	Disp:	Sig:
Podofilox 0.5% gel	Condylox	3.5 g	Apply to lesions with a cotton-tip applicator b.i.d. then withdraw for the next 4 days. Repeat cycle weekly up to 4 times, if needed.
Cidofovir 1% cream or 3% gel	Vistide	5 g	Apply and rub into lesions with a cotton-tip applicator on q.o.d. for 1 week.
Imiquimod 5% cream	Aldara	5 g	Apply to lesions with a cotton-tip applicator on a once daily basis for 2 weeks.

Vaccines

To Prevent Recurrent Herpes Zoster (Shingles) Infections

Live, attenuated varicella-zoster virus (VZV; shingles) vaccine	Zostavax	Vial	Single dose subcutaneous injection. For individuals 60 years of age and older.
---	----------	------	--

To Prevent Oral Human Papillomavirus Infections and Related Disease

Quadrivalent HPV vaccine (low-risk HPV types 6 and 11, and high-risk HPV types 16 and 18)	Gardasil	Vial	3 injections given in 1 year at month 0, month 2, and month 6.
---	----------	------	--

TOPICAL ORAL ANESTHETIC AGENTS

To Manage and Provide Short-Term Relief of Chronic Pain Associated with Mucosal Ulcerations

<i>Generic Name and Concentration</i>	<i>Trade Name</i>	<i>Disp:</i>	<i>Sig:</i>
Diphenhydramine HCl 12.5 mg/5 mL elixir	Benadryl	4 fluid oz.	Rinse with 1 tablespoonful a.c. and p.r.n. for pain.
Benadryl elixir 12.5 mg/5 mL and Kaopectate (Upjohn) 50% mixture by volume		4 fluid oz of each; mix equal parts	Rinse with 1 tablespoonful for 2 minutes a.c. and p.r.n. for pain
Lidocaine HCl 2% viscous solution HCl	Xylocaine	4 fluid oz.	Rinse with 1 tablespoonful a.c. and p.r.n. for pain.
Orabase with Benzocaine (OTC)		5 or 15 g	Apply to affected area a.c. and p.r.n. for pain.
Dyclonine 0.5% or 1%		30 mL	Apply to affected area a.c. and p.r.n. for pain.
Lidocaine 4% gel	Topicaïne-4	10 or 30 g	Apply to affected area a.c. and p.r.n. for pain.

ANTIANXIETY AGENTS

To Manage and Provide Short-Term Relief of the Symptoms of Anxiety associated with Dental Procedures

<i>Generic Name and Concentration</i>	<i>Trade Name</i>	<i>Disp:</i>	<i>Sig:</i>
Alprazolam 0.25 mg	Xanax	20	Take 1 tablet in evening before bed and 1 tablet 1 hour before appointment.*
Chlordiazepoxide 10 mg	Librium	20	Take 1 tablet in evening before bed and 1 tablet 1 hour before appointment.*
Triazolam 0.25 mg	Halcion	20	Take 1 tablet 1 hour before dental appointments.*
Diazepam 5 mg	Valium	20	Take 1 tablet in evening before bed and 1 tablet 1 hour before appointment.*
Oxazepam 10 mg	Serax	20	Take 1 tablet in evening before bed and 1 tablet 1 hour before appointment.*
Buspirone 5 mg	BuSpar	20	Take 1 tablet q.d. or b.i.d.
Lorazepam 0.5 mg	Ativan	20	Take 1 tablet q.d. or b.i.d.
Hydroxyzine 25 mg	Atarax, Vistaril	20	Take 2 capsules in evening before bed and 2 capsules 1 hour before appointment. Note: Dose of child (30–60 lb.) is half the adult dose.*
Hydroxyzine syrup 10 mg/5 mL	Atarax	50 or 100 mL	Take 2 teaspoonfuls 1 hour before dental appointment.
Diphenhydramine 25 mg	Benadryl	25	Take 1 tablet q.i.d.

Note: Use lower doses of these drugs in the elderly and in patients who have kidney or liver impairment.

*Caution: patients should not drive themselves to or from the appointment.

FLUORIDE THERAPY

To Prevent Dental Caries in Susceptible Patients

Generic Name and Concentration	Trade Name	Disp:	Sig:
Stannous fluoride 0.4%	Gel-Kam	4.3 fluid oz.	Apply 5-10 drops in a carrier and place carrier on teeth daily for 5 minutes
Sodium fluoride 0.11 %	Luride	60 mL bottle with dropper	6 months to 3 years of age: apply one-half dropperful (0.5 mL) daily to mouth of child 3 to 6 years: apply one dropperful daily in mouth of child <i>Consider using when drinking water fluoride content is less than 0.3 ppm fluoride.</i>
1.1 % neutral sodium fluoride (5000 ppm)	Preident, ControlRx, Neutracare Home Topical	2 oz.	Apply 5 drops in a custom made tray and place tray over teeth daily for 5 minutes
Fluoride varnish (22,600 ppm)	CavityShield, Duraphat, VarnishAmerica	1 oz tube	Apply to susceptible tooth surfaces using cotton tip in dental office every 6 months

MUCOSAL ULCER MEDICATIONS

Steroids

To Manage Mucosal Ulcers and Erosions associated with Immunological and Skin Disorders

Generic Name and Concentration	Trade Name	Disp:	Sig:
Medium Potency Steroids			
Triamcinolone acetonide 0.1 % ointment	Kenalog in Orabase	5 g tube	Apply to ulcerated area p.c. and h.s.
Betamethasone valerate 0.1 % ointment	Valisone ointment	15 g or 30 g tube	Apply to ulcerated area p.c. and h.s.
Betamethasone syrup 0.1 %	Celestone	50 mL	Rinse with 1 teaspoonful q.i.d., p.c., and h.s.
Triamcinolone 0.1 % ointment	Kenalog-40	5 mL injectable vials	Mix 1 mL of steroid with 0.5 mL of anesthetic. Inject 0.25 mL at 4 locations around border of ulcer. Total 1 mL injected.
High Potency Steroids			
Augmented betamethasone 0.05 % ointment	Diprolene ointment	15 or 30 g tube	Apply to affected area p.c. and h.s.
Fluocinonide 0.05 % ointment	Lidex ointment	15 or 30 g tube	Apply to mouth sore p.c. and h.s.
Dexamethasone elixir 0.5 %	Decadron	100 mL bottle	Rinse with 1 teaspoonful q.i.d. for 2 minutes, then expectorate.
Very High Potency Steroids			
Dexamethasone elixir 0.75 %	Decadron	100 mL bottle	Rinse with 1 teaspoonful q.i.d. for 2 minutes, then swallow.
Clobetasol 0.05 % ointment	Temovate ointment	15 or 30 g tube	Apply to affected area q.i.d.
Halobetasol propionate 0.05 % ointment	Ultravate ointment	15 or 30 g tube	Apply to affected area q.i.d.
Methylprednisolone	Medrol 4 mg Dosepak 21s	1 Dosepak (21 tabs)	Take graduated daily doses according to the manufacturer's directions listed on the Dosepak.
Prednisone 10 mg		20	Take 4 tablet in the a.m. Continue daily for 4 d.

*High potency and very high potency steroids should be avoided in patients with gastrointestinal ulcers, diabetes, hematologic malignancy, and hepatitis and in women who are pregnant or nursing.

More than 2 weeks of therapy of very high potency steroids may affect normal cortisol production and increase risk of candidiasis

Immunomodulating Drugs

<i>Generic Name and Concentration</i>	<i>Trade Name</i>	<i>Disp:</i>	<i>Sig:</i>
Azathioprine 50 mg	Imuran	60	Take 1 tablet b.i.d.
Imuran can be prescribed to reduce the dose of prednisone.			
Tacrolimus 0.1 %	Protopic	30 g	Apply to affected area q.i.d.
Dapsone 25 mg	Dapsone	60	Take 1 tablet t.i.d., 2 t.i.d., 3 t.i.d., 4 daily thereafter

Dapsone requires checking complete blood count and liver function every month for 3 months

Other Agents Ulcer Coating Agents

Active ingredient: 2-alkyl cyanoacrylate	Orabase Soothe-n-Seal	1 box of 10 applicators	Drip 2 drops then soak applicator tip with solution, and apply as a covering to ulcer.
<i>Note:</i> can be applied over steroid ointment applications.			
Diphenhydramine HCl with Kaopectate	Benadryl Syrup (Mix 50/50) with Kaopectate	8 oz	Rinse 1 tablespoonful in mouth for 1 minute then spit. Repeat as needed to relieve pain.

Cauterizer (for aphthous)

Active ingredient: Sulfuric acid and phenolic solution	Debacterol	1 tube	Break tube to open, touch saturated cotton tip to ulcer for 20 seconds. Advise patient it will hurt (burn).
--	------------	--------	---

NUTRIENT DEFICIENCY THERAPY

To Replace Deficient Nutrients Necessary for Homeostasis

<i>Generic Name and Concentration</i>	<i>Trade Name</i>	<i>Disp:</i>	<i>Sig:</i>
Ferrous sulfate 250 mg	Iron	100	Take 1 tablet q.d. for 1 month, then reassess patient's hemoglobin level.
Folic acid 0.4 mg	Folate	30	Take 1 tablet q.d. for 1 month, then reassess patient's hemoglobin level.
Cyanocobalamin	Vitamin B12	10 mL vial	Inject 0.1 to 1 mL IM in deltoid; reassess patient's symptoms and blood profile monthly.
Water-soluble bioflavonoids 200 mg with ascorbic acid 200 mg	Peridin-C 400 mg	100	Take 1 tablet t.i.d.

SALIVA SUBSTITUTE

To Relieve Dry Mouth

Generic Name and Concentration	Trade Name	Disp:	Sig:
Carboxymethyl cellulose 0.5% aqueous solution	Saliva substitute	120 mL	Use as a rinse p.r.n.
Carboxymethyl cellulose	Moi-Stir ^a , Orex, Sage Moist Plus, Salivart ^b , Xero-Lube, MouthKote, Salix		Use as a rinse p.r.n.
Hydroxyethylcellulose, xylitol, citric acid	Optimoist	9 mL	Spray in mouth p.r.n. for dry mouth
Glucose oxidase and lactoperoxidase	Biotene	120 mL	Rinse in mouth for 1 minute p.r.n. for oral dryness.
Pilocarpine 5 mg	Salagen	100	Take 1 tablet t.i.d. or q.i.d.
<i>Caution:</i> contraindicated with glaucoma; precautions (eye, heart, obstructive lung diseases)			
Cevimeline HCl 30 mg	Evoxac	100	Take 1 tablet t.i.d. p.r.n. for oral dryness.
<i>Caution:</i> contraindicated with glaucoma; precautions (eye, heart, obstructive lung diseases)			

^a120 mL with pump spray

^b75 mL with pump spray

SEDATIVE/HYPNOTICS

To Produce a Sleeplike State for the Effective Dental Management of the Patient

<i>Generic Name and Concentration</i>	<i>Trade Name</i>	<i>Disp:</i>	<i>Sig:</i>
Triazolam 0.25 mg	Halcion	30	Take 1 tablet h.s.
Flurazepam 15 mg	Dalmane	30	Take 1 tablet h.s.
Temazepam 15 mg	Restoril	30	Take 1 tablet h.s.
Chloral hydrate 500 mg/5 mL	Noctec	1 pint	Take 1 teaspoonful before bedtime or 30 minutes before surgery.

Caution: patients should not drive themselves to or from appointment.

TOBACCO CESSATION

To Aid the Patient in Quitting the Use of Tobacco

First: Ask about your patient's tobacco use
Advise them to quit
Assess their readiness to quit
Assist if ready to quit (medications, substitutes and counseling)
Arrange for follow-up care

Generic Name and Concentration	Trade Name	Disp:	Sig:
Varenicline 1 mg	Chantix	60	Take 1/2 tablet per day for 3 days before quit date, then 1/2 tablet b.i.d. (>8 hours apart) for 4 days, then 1 tablet b.i.d. (>8 hours apart) for 12 weeks.
Wellbutrin SR 150 mg	Zyban	60	Take 1 tablet per day for 3 days before quit date, then 2 tablets per day (>8 hours apart) for 8 to 12 weeks.

GUIDE TO DIAGNOSIS AND MANAGEMENT OF COMMON ORAL LESIONS

White Lesions

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Fordyce's granules	Any	M-F	Any	Ectopic sebaceous glands	Whitish yellow granules clustered in plaques located on buccal mucosa (bilaterally), labial mucosa, retro-molar pad, lip, attached gingiva, tongue, and frenum. Lesions are nontender and rough to palpation and do not rub off. Onset after puberty; persists for life.	None required.
Linea alba buccalis	Any	M-F	Any	A type of frictional keratosis caused by persistent rubbing of buccal mucosa with interdigitating maxillary and mandibular teeth	White wavy line of varying length located on buccal mucosa, bilaterally. Lesions are nontender and smooth to palpation and do not rub off. Variable onset; persists with oral habits.	Eliminate bruxism and clenching.
Leukoedema	Any	M-F	Meloderms	Unknown	Grayish white patch of variable size located on buccal mucosa (bilaterally), labial mucosa, and soft palate. Lesions are nontender and smooth to palpation and disappear when the mucosa is stretched. Leukoedema becomes more evident with increasing age.	None required.
Morsicatio biccarum	Any	M-F	Any	Chronic irritation caused by cheek biting	Asymmetric white plaque located on buccal mucosa and labial mucosa, often bilaterally. Lesions are nontender and rough to palpation and peel slightly when rubbed. Variable onset; persists with cheek-biting or lip-biting habit.	Eliminate cheek-biting or lip-chewing habit.
White sponge nevus	Any	M-F	Any	Autosomal dominant condition (mutation in keratin 4 or 13 genes) that results in defect in epithelial maturation and exfoliation	Solitary or confluent raised white plaques that may appear on buccal mucosa, labial mucosa, alveolar ridge, floor of the mouth, or soft palate. Lesions are nontender and rough to palpation and do not rub off. Onset at birth; persists for life.	None required.

(continued)

White Lesions (*Continued*)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Traumatic white lesions	Any	M-F	Any	Acute chemical or physical trauma resulting in epithelial sloughing	White surface slough (eschar) usually located on the less-keratinized alveolar mucosa. Palate common for food burns. Lesions are tender to palpation and rub off, leaving a raw or bleeding surface. Onset within hours of trauma; regression in 1–2 weeks.	Eliminate irritant; use topical anesthetics and analgesics.
Leukoplakia	45–65	2:1 (M:F)	Any	Frequently related to tobacco or alcohol use	White patch that varies in size, homogeneity, and texture. High-risk locations include floor of the mouth, ventral tongue, lateral tongue, and uvulopalatal complex. Lesions do not rub off and usually are nontender. Onset occurs after prolonged contact with an inducing agent; persists as long as the inducing agent is present.	Biopsy and histologic examination. Close follow-up mandatory.
Cigarette keratosis	Elderly	M	Any	Heat and smoke from frequent smoking of nonfiltered cigarettes	White pebbly circular patches located on upper and lower lips (kissing lesions). Keratoses are firm and nontender and do not rub off. Onset in conjunction with a prolonged cigarette smoking habit; persists with habit.	Use filtered cigarettes or stop smoking. Biopsy if lesion changes in color, or becomes ulcerated or indurated.
Nicotine stomatitis	40–70	M	Any	Epithelial reaction to tobacco smoke or heat	White cobblestoned papules located on hard palate, excluding the anterior third. Papules have red centers, are nontender, and do not rub off. Onset varies according to degree of smoking; lesions are longstanding.	Stop pipe or cigar smoking, or reverse smoking habit.

(continued)

White Lesions (Continued)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Snuff dipper's patch	Teenagers and adult	M	Any	Chronic irritation of smokeless tobacco products	Corrugated whitish yellow patch located on mucobuc- cal fold, most prominent unilaterally. Patch is rough and nontender and does not rub off. Long-standing habit precedes lesion; patch persists with continuation of habit.	Discontinue tobacco use. Biopsy if color changes or if lesion becomes ulcerated or indurated.
Verrucous carcinoma	>60	M	Any	Neoplastic changes induced by long- term use of tobacco, smokeless tobacco, and human papillo- mavirus infection	Papulonodular whitish red mass located on buccal mucosa, alveolar ridge, gin- giva. Lesion is firm, non- tender, and rough to palpa- tion and does not rub off. Long-standing tobacco habit precedes onset; human papillomavirus pres- ent in 30%; lesion enlarges unless treated.	Biopsy to confirm diagnosis, then surgi- cal exci- sion. AVOID RADIATION THERAPY
Squamous cell carcinoma (see "Red and Red- White Lesions")						

Red Lesions

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Purpura	Any	M-F	Any	Rupture of blood vessel caused by trauma or vascular abnormality	Red spot or patch consist- ing of extravasated blood that develops soon after trauma. Lesions do not blanch on diascopy and size varies (petechiae < ecchymosis < hematoma). Petechiae are common on soft palate; other purpura typically occur on buccal or labial mucosa, depending on site at which blood pools. Lesions fade away.	Eliminate underlying cause.

(continued)

Red Lesions (Continued)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Varicosity	>55	F	Any	Dilated veins caused by loss of elasticity	Reddish purple papule or nodule located on ventral tongue, lip, or labial mucosa. Lesions are asymptomatic and blanch on diascopy. Varicosities increase in size and number with increasing age and are persistent.	None necessary. Surgery for aesthetics.
Thrombus	>30	M-F	Any	Blood clot caused by stagnating blood or clotting abnormality	Red to blue-purple nodule located on labial mucosa, lip, or tongue. Lesions are firm and may be tender to palpation. Onset after traumatic bleeding; lesion is negative on diascopy and persists until treatment. Thrombi sometime spontaneously regress.	Surgical removal and histologic examination, if persistent or symptomatic.
Hemangioma	Child-adolescent	F	Any	Congenital abnormality resulting in a network of blood vessels in bone or soft tissue	Two types described: capillary and cavernous. Red to purple, soft, smooth-surfaced, or multinodular exophytic mass located on dorsal tongue, buccal mucosa, or gingiva. Lesions are positive on diascopy. Develops early in life, persists until treated. Hemangiomas sometimes spontaneously regress.	No treatment is necessary if present since youth, no functional disability, and no changes in size, shape, or color. Otherwise, surgery or histologic examination.
Hereditary hemorrhagic telangiectasia	Post-puberty	M-F	Any	Autosomal dominant condition associated with multiple dilated end capillaries that results from a defect in a transmembrane protein (endoglin)—a component of the receptor complex for transforming growth factor beta (TGF- β)	Multifocal red macules located on palms, fingers, nail beds, face, neck, conjunctiva, nasal septum, lips, tongue, hard palate, and gingiva. Lesions are present at birth, become more visible at puberty, and increase in number with age. Telangiectasias lack central pulsation and blanch on diascopy; if they rupture, severe bleeding may result.	Avoid trauma and intubation. Monitor for hemorrhage or anemia.

(continued)

Red Lesions (Continued)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Sturge-Weber syndrome	Birth	M-F	Any	Nonhereditary congenital disorder that results in multiple venous angiomas at various anatomic sites	Syndrome associated with seizures, mental deficits, gyriform brain calcifications, and a red to purple flat or slightly raised facial hemangioma. Vascular lesion often affects lips, labial or buccal mucosa, and gingiva along branches of trigeminal nerve, stops at midline. Abnormal oral enlargements may be concurrent.	None required. Elective surgery for aesthetics.
Kaposi sarcoma	20–45* and >60	M	Jewish, Mediterranean, or *HIV-infected	Associated with human herpesvirus type 8, a virus capable of promoting angiogenesis	Asymptomatic red macule of mucocutaneous structures that enlarges and becomes raised, then darkens in color. Advanced lesions are red-blue-violet nodules that ulcerate and cause pain. The hard palate, gingiva, and buccal mucosa are the most common oral locations.	Palliative, consisting of radiation therapy, laser surgery, chemotherapy, sclerosing agents, or a combination thereof.
Erythroplakia	>50	M>F	Any	Increased vascularity associated with carcinogen-induced epithelial changes and inflammation	Red patch of variable size located on any oral mucosal site. High-risk areas include floor of the mouth, soft palate-retromolar trigone, and lateral border of tongue. Erythroplakias do not rub off and are usually asymptomatic. Lesions develop after prolonged contact with carcinogens or human papillomavirus; duration varies. Regression is rare.	Biopsy and histologic examination. Close follow-up.

(continued)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Erythroleukoplakia and speckled erythroplakia	>50	M>F	Any	Increased vascularity associated with carcinogen-induced and <i>Candida</i> -induced epithelial changes and inflammation	Red patch with multiple foci of white. Nontender, do not rub off, often superficially infected with <i>Candida</i> organisms. Common locations include lateral tongue, buccal mucosa, soft palate, and floor of the mouth. Onset after prolonged exposure to carcinogens or human papillomavirus. Regression unlikely even if inducing agent is removed.	Biopsy and histologic examination. Examine for candidiasis. Close follow-up.
Squamous cell carcinoma	>50	2:1 M:F	Any	Prolonged exposure to carcinogens (tobacco, alcohol, or human papillomavirus) and decreased immune surveillance	Red or red and white lesion or ulcer commonly located on lateral tongue, ventral tongue, oropharynx, floor of the mouth, gingiva, buccal mucosa, or lip. Carcinoma often asymptomatic until it becomes large, indurated, or ulcerated. Onset after prolonged exposure to carcinogens. Persistence results in metastasis, usually apparent as painless, firm, matted, fixed lymph nodes.	Biopsy and histologic examination. Complete surgical removal, radiation therapy, or chemotherapy. Close follow-up.
Lichen planus	>40	F	Any	T-cell infiltration and cytokine-induced changes in epithelium; exact inducer unknown	Purple, polygonal, pruritic papules on flexor surfaces of skin; fingernails sometimes affected. Intraoral lesions are often symptomatic and consist of white linear papules, reddish patches, and ulcerated regions of mucosa. Affected surfaces are often bilateral. Most common locations: buccal mucosa, tongue, lips, palate, gingiva, and the floor of the mouth. Lesions develop with stress and liver disease; they persist for many years with periods of remission and exacerbation.	Rest, anxiolytic agents, topical corticosteroids. Evaluate for liver disease; close follow-up for occasional malignant transformation in the erosive type.

(continued)

Red and Red-White Lesions (*Continued*)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Electro-galvanic white lesion	>30	F	Any	Metal antigen of a dental restoration that induces a hyperimmune T-cell response	Reddish white patches that resemble lichen planus located on buccal mucosa adjacent to metallic restorations. Lesions do not rub off and are usually tender or cause a burning sensation. Onset after weeks to years of exposure to metallic restoration; duration varies depending on the persistence of the allergen.	Replace metallic restoration or clasp that is causing the hypersensitive response.
Lupus erythematosus	>35	F	Any	Autoantibodies (antinuclear) that attack normal cells leading to perivascular inflammation	Reddish butterfly rash on bridge of nose. Maculopapular eruption with hyperkeratotic periphery and atrophic center. Affected areas may involve the lower lip, buccal mucosa, tongue, and palate. Intraoral lesions invariably have red and white radiating lines emanating from the lesion. Lesions do not rub off but are tender to palpation. Lesions often develop after short-term sun exposure. Lesions persist and require drug treatment.	Topical and systemic steroids; antimalarial agents in conjunction with adequate medical treatment.
Lichenoid and lupuslike drug eruption	Adult	M-F	Any	Drug molecules act as allergens or haptens that stimulate immune reaction	Red-white patches that resemble lichen planus and lupus. The lesions are often atrophic or ulcerated centrally. Buccal mucosa, bilaterally, is the most common site. Onset varies and may be weeks or years after an allergenic medication is begun. Regression occurs when the offending drug is eliminated.	Withdraw offending drug and substitute medication.

(continued)

Red and Red-White Lesions (Continued)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Candidiasis	Newborns, adults	M-F	Any	Opportunistic infection with <i>Candida</i> species (most commonly <i>C. albicans</i>)	Variable appearance; white curds, red patches, white patches with red margins. Any oral soft tissue site is susceptible, but the attached gingiva is rarely affected. Onset often coincides with neutropenia, immune suppression and frequent use of steroids or antibiotics. Lesions persist until adequate antifungal therapy is provided.	Antifungal therapy. Eliminate sucrose from diet. Medical control of diabetes, endocrinopathy, and immunosuppression.

Pigmented Lesions

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Melanoplakia	Any	M-F	Melanoderms	Melanin deposition in the basal layer of the mucosa and lamina propria	Generalized constant dark patch located on attached gingiva and buccal mucosa. Pigmentation varies from light brown to dark brown and is often diffuse, curvilinear, and asymptomatic and does not rub off. Melanoplakia is present at birth and persists for life.	None required.
Tattoo	Teenagers, adults	M-F	Any	Implantation of dye or metal in mucosa	Amalgam tattoo is the most common type of intraoral tattoo. Appears as a blue-black macule on gingiva, edentulous ridge, vestibule, palate, or buccal mucosa. Radiographs may demonstrate radiopaque foci. Lesions are asymptomatic, do not blanch, and persist for life.	None required.
Ephelis	Any	M-F	Light-skinned persons	Deposition of melanin triggered by exposure to sunlight	Light to dark brown macule that appears on facial skin, extremities, or lip after sun exposure. Ephelides are initially small but may enlarge and coalesce. Lesions are nontender and do not blanch or rub off.	None required.

(continued)

Pigmented Lesions (*Continued*)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Smoker's melanosis	Older adult	M-F	Any	Accumulation of melanocytes and deposition of melanin in epithelium associated with tobacco smoking	Diffuse brown patch with diameter of several centimeters, usually on posterior buccal mucosa and soft palate. History of heavy tobacco smoking precedes development of the lesion. Features may decrease with discontinuation of the habit. Melanosis is asymptomatic and nonpalpable.	Diminish or stop smoking.
Oral melanotic macule	24–45	Slight male predilection	Any	Focal deposition of melanin along the basal layer, usually after trauma	Asymptomatic brown to black macule usually located on lower lip near midline; also occurs on palate, buccal mucosa, and gingiva. Onset is postinflammatory, and the lesion persists until treatment.	Biopsy and histologic examination to rule out other similar-appearing pigmented lesions.
Nevus	Any	F	Any	Accumulation of nevus cells in a distinct location, probably controlled by genetic factors	Nevi vary greatly in appearance. They may be pink, blue, brown, or black but do not blanch on diascopy. Usually appear as a bluish or brownish smooth-surfaced papule located on the palate. Other common sites include the buccal mucosa, face, neck, and trunk. Many lesions are present at birth. They increase in size and number with increased age.	Excisional biopsy and histologic examination.
Melanoma	25–60	M	White persons, especially light-skinned persons	Malignant neoplasm of melanocytes associated with chronic ultraviolet light exposure; unknown for oral melanomas	Painless, slightly raised plaque or patch that has many colors, especially foci of brown, black, gray, or red. Ill-defined margins, satellite lesions, and inflammatory borders are characteristic. Usually located on the maxillary alveolar ridge, palate, anterior gingiva, and labial mucosa. Thirty percent arise from pre-existing pigmentations. A recent change in size, shape, or color is particularly ominous.	Excisional biopsy, surgical removal, and referral for complete medical workup to rule out metastasis.

(continued)

Pigmented Lesions (Continued)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Peutz-Jeghers syndrome	Child, young adult	M-F	Any	Autosomal dominant condition probably caused by a germline mutation in the <i>LKB1</i> gene (19p13.3)	Multiple, asymptomatic melanotic oval macules, prominently located on the skin of the palmar or plantar surfaces of the hands and feet, around the eyes, nose, mouth, lips, and perineum. In the mouth, brown discolorations occur on the buccal mucosa, labial mucosa, and gingiva. Lesions do not increase in size, but cutaneous lesions often fade with age; mucosal pigmentation persists for life. Colicky intestinal symptoms are probable.	Oral: none required. Gastrointestinal evaluation and genetic counseling.
Addison disease	Adult	M-F	Any	Adrenal hypofunction	Diffuse intraoral hypermelanotic patches occurring in conjunction with bronzing of the skin, especially of the knuckles, elbows, and palmar creases. Patches are nontender and nonraised and vary in shape. The buccal mucosa and gingiva are most commonly affected. Onset of the disorder is insidious and associated with adrenal gland hypofunction. Patient may report gastrointestinal symptoms and fatigue.	Systemic corticosteroids and mineralocorticoids.
Heavy metal pigmentation	Adult	M-F	Any	Prolonged exposure (vapors or ingestion) to metals (arsenic, bismuth, mercury, silver, or lead)	Blue-black linear pigmentation of marginal gingiva, prominently viewed along anterior gingiva. Spotty gray macules may be apparent on buccal mucosa. Neuralgic symptoms, headache, and hypersalivation are common. Argyria: blue-gray skin pigmentation, especially in sun-exposed areas.	Terminate exposure to heavy metal; provide medical referral. Oral lesions require no treatment, but may be permanent.

(continued)

Papules and Nodules

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Retrocuspid papilla	Child, young adult	M-F	Any	Developmental anomaly of connective tissue	Smooth-surfaced pink papule, 1–4 mm in diameter, located on the lingual attached gingiva apical to the marginal gingiva of the mandibular cuspids. These papules appear early in life, are often found bilaterally, and regress as the patient ages. The retrocuspid papilla is firm to palpation, asymptomatic, and non-hemorrhagic.	None required.
Lympho-epithelial cyst	Child, young adult	M-F	Any	Epithelium entrapped in lymphoid tissue that undergoes cystic transformation	Well-circumscribed, soft, fluctuant yellowish swelling that ranges in size from a few millimeters to 1 cm. Common locations for this nontender cyst include the lateral neck just anterior to the sternocleidomastoid muscle, floor of the mouth, lingual frenum, and ventral tongue. Lesion develops during childhood or adolescence and persists until treatment.	Excisional biopsy and histologic examination.
Torus, exostosis, and osteoma	Adult	F	Any	Unknown hereditary factors	Torus: bony hard nodule or multinodular mass located on the palate at the midline, or mandibular lingual alveolar ridge. Exostosis: bony hard nodule, often multiple, located on buccal or labial alveolar ridge. Osteoma: bony hard nodule located adjacent to the jaws, often embedded in soft tissue. All three types are firm, painless (unless traumatized), slow growing, and long-standing. Osteomas have the greatest growth potential.	Torus and exostosis: none required unless functional problems arise. Osteomas plus impacted and supernumerary teeth: gastrointestinal evaluation to rule out Gardner syndrome (gene defect on chromosome 5). If test results are positive, then gastrointestinal surgery and genetic counseling.

(continued)

Papules and Nodules (*Continued*)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Fibroma	Adult	M-F	Any	Usually a chronic irritant that produces a reactive hyperplasia of connective tissue	Irritation fibroma is a smooth-surfaced, pink, firm, symmetric papule or nodule that arises at a site of chronic irritation, such as the buccal mucosa, labial mucosa, and tongue. The gingiva is the common location for the peripheral odontogenic fibroma. Both lesions have sessile bases and are non-tender and nonhemorrhagic.	Excisional biopsy and histologic examination.
Lipoma	>30	M-F	Any	Unknown genetic mutation possibly involving dysfunction proteins that regulate chromatin structure and function	Well-circumscribed, smooth-surfaced, dome-shaped, yellowish to pink nodule commonly located on buccal mucosa, lip, tongue, floor of the mouth, soft palate, or mucobuccal fold. Lesion is slightly doughy on palpation and grows slowly.	Excisional biopsy and histologic examination.
Lipofibroma	>30	M-F	Any	Unknown genetic mutation	Well-circumscribed, smooth-surfaced, dome-shaped, pinkish nodule commonly located on buccal or labial mucosa. Lesion is painless, movable, and rather firm. Slow growth and persistence are characteristic.	Excisional biopsy and histologic examination.
Traumatic neuroma	>25	M-F	Any	Trauma to large nerve that results in abnormal healing and aberrant neural conduction	Small, slightly raised, firm, pressure-sensitive papule that is commonly located in the mandibular mucobuccal fold near the mental foramen, facial to mandibular incisors, lingual retromolar regions, and ventral tongue. Visualization of the lesion may be difficult if the neuroma is subjacent to normal-appearing mucosa. Palpation elicits an electric shock sensation. Onset occurs after trauma; lesions persist until treated.	Excisional biopsy and histologic examination; if lesion recurs, corticosteroid injections may be effective.

(*continued*)

Papules and Nodules (*Continued*)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Neuro-fibroma	Childhood	M-F	Any	Gene dysregulation resulting in uncontrolled growth of peripheral nerves and neural sheaths	Firm pink nodules that are often deep-seated. These tumors are located in skin, bones, buccal mucosa, tongue, or lips. Lesions are nontender but movable. Continued enlargement can lead to deformity. Multiple lesions and skin pigmentations are associated with von Recklinghausen disease (neurofibromatosis).	Excisional biopsy, histologic examination, and follow-up for malignant transformation in cases of neurofibromatosis.
Papilloma	20–40	M	Any	Epithelial growth induced by chronic human papillomavirus infection (primarily types 6 and 11)	Small, pink, pebbly, slow-growing papule located on uvula, soft palate, tongue, frenum, lips, buccal mucosa, or gingiva. The base is pedunculated and well circumscribed, whereas the surface is most often rough to palpation.	Excisional biopsy and histologic examination.
Verruca vulgaris	Child, young adult	M-F	Any	Epithelial growth induced by chronic human papillomavirus infection (primarily types 2 and 4)	Rough, whitish pink papule located on the skin of the hands and periorally on the lips, labial and buccal mucosa, and attached gingiva. Lesions are slow growing and have a sessile base. Verrucae may regress spontaneously or spread to adjacent mucocutaneous surfaces.	Excisional biopsy and histologic examination.
Condyloma acuminatum	20–45	M	Any	Epithelial growth induced by chronic human papillomavirus infection (primarily types 6 and 11)	Small, pink-to-dirty gray papule with rough, papillary surface that resembles a cauliflower. Base of condyloma is sessile, and the borders are raised and rounded. Lesions occur in multiples, and onset rapidly occurs after inoculation from affected sexual partner. Most common location is genitalia, labial mucosa, labial commissure, attached gingiva, and soft palate. Lesions can spread and coalesce into extensive clusters.	Excisional biopsy and histologic examination.

(continued)

Papules and Nodules (*Continued*)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Lymph-angioma	Child, adolescent	M-F	Any	Congenital hamartoma of lymphatic channels	Soft, compressible, pinkish white swelling that may be superficial or deep-seated. Superficial lesions resemble papillomas; deep-seated lesions cause diffuse enlargement. Lymphangiomas may occur in neck (cystic hygroma), dorsal or lateral tongue, lip, or labial mucosa. Long-standing lesions can cause functional problems or regress spontaneously.	Surgical excision; depending on size and location, surgery may require general anesthesia.

Vesiculobullous Diseases

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Primary herpetic gingivostomatitis	Infant, child, young adult	M-F	Any	Primary herpes simplex virus (type 1) infection of oral epithelium; infection with type 2 is less likely	Multiple vesicles that rupture, coalesce, and form ulcers of the lip, buccal and labial mucosa, gingiva, palate, and tongue. Ulcers are painful and initially are small and yellow, and have red inflammatory borders. Onset is rapid, several days after contact with person harboring the virus. Lesions persist for 12–20 days.	Fluids, antivirals, antipyretics; antibiotics to prevent secondary infection, oral anesthetic rinses, analgesics.
Recurrent herpes simplex	Teenagers and adults	M-F	Any	Recrudescence of herpes simplex virus from sensory neuron, resulting in infection of orofacial epithelium, trauma and stress associated	Multiple small vesicles that rupture and ulcerate. Lesions occur repeatedly at same site: usually the lip, hard palate, and attached gingiva. Onset is rapid and is preceded by prodromal burning or tingling. Lesions last 5–12 days and heal spontaneously.	Antivirals (acyclovir, famciclovir, valacyclovir), bioflavonoids, sunscreens (lip), lysine.

(*continued*)

Vesiculobullous Diseases (*Continued*)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Herpangina	Child, young adult	M-F	Any	Coxsackie virus (types A1–6, A8, A10, A22, B3) and echovirus infection of oropharynx	Light gray vesicles that rupture and form many discrete shallow ulcers. Lesions have erythematous border and are limited to the anterior pillars, soft palate, uvula, and tonsils. Pharyngitis, headache, fever, and lymphadenitis are often concurrent. Lesions heal spontaneously in 1–2 weeks.	Palliative; vesicles heal spontaneously.
Chickenpox	Child	M-F	Any	Primary varicella zoster virus infection	Vesicles on skin and face that, after rupturing, resemble a dewdrop. Ulcers may be seen on soft palate, buccal mucosa, and mucobuccal fold. Skin lesions crust over and heal without scar formation. Condition often accompanied by chills, fever, nasopharyngitis, and malaise. Spontaneous healing occurs in 7–10 days.	Vaccination; palliative; vesicles heal spontaneously. Avoid scratching to limit scar formation.
Herpes zoster	>55, >35 in HIV- positive	M-F	Any	Recrudescence of varicella zoster virus from sensory neuron, resulting in infection of epithelium	Unilateral vesicular and pustular eruptions that develop over 1–3 days. Lesions occur along dermatomes and especially along the trigeminal nerve tract. Lesions are vesicular, ulcerative, and intensely painful and commonly affect the lip, tongue, and buccal mucosa extending up to the midline. Neuralgia may persist after healing.	Vaccination; palliative; lesions heal spontaneously. Antivirals (e.g., Famciclovir in severe cases or immunosuppressed patients.
Hand-foot-and-mouth disease	Child, young adult	M-F	Any	Coxsackie virus (types A5, A9, A10, B2, B5) infection	Crops of multiple small yellowish ulcers that occur on palms and soles of hands and feet. The tongue, hard palate, and buccal and labial mucosa are affected. Total number of lesions may approach 100. Healing occurs spontaneously in about 10 days.	Palliative; ulcers heal spontaneously.

(continued)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Allergic reactions immediate	Any	M-F	Any	Immediate immunologic reaction involving an allergen, IgE, and the release of histamine from mast cells	Red swellings or wheals that occur periorally or on lips, buccal mucosa, gingiva, lips, and tongue. Contact with allergen usually precedes episode by a few minutes to hours. Warmth, tenseness, and itchiness are concurrent. Lesions regress if the allergen is withdrawn.	Remove allergen; antihistamines.
Allergic reactions delayed	Any	M-F	Any	Immunologic reaction after 24 to 48 hours involving an allergen, T cells, and a cytotoxic reaction	Itchy erythematous papules or vesicles that may eventually ulcerate. May occur on any cutaneous or mucocutaneous surface. The lips, gingiva, alveolar mucosa, tongue, and palate are affected. Erythema develops slowly over 24–48 hours. Fissuring and ulceration may result.	Remove allergen; corticosteroids.
Erythema multiforme	Young adult	M	Any	Complement-mediated and white blood cell-mediated cytopathic effects provoked by pathogens and drug allergens	Skin: target lesions. Oral: hemorrhagic crust of the lips, painful ulcerations of the tongue, buccal mucosa, and palate. Attached gingiva rarely affected. Headache, low-grade fever, and previous respiratory infection often precede lesions.	Topical analgesics, antivirals, antipyretic agents, fluids, corticosteroids, antibiotics to prevent secondary infection.
Stevens-Johnson syndrome	Child, young adult	Slight preference for males	Any	Complement-mediated and white blood cell-mediated cytopathic effects provoked by pathogens and drug allergens occurring at more than one location	Skin: target lesions. Eye: conjunctivitis. Genital: balanitis. Oral: hemorrhagic crust of the lips, painful ulcerations, and weeping bullae of the tongue and buccal mucosa. Attached gingiva rarely affected. Stevens-Johnson syndrome is the fulminant form of erythema multiforme. Eating and swallowing are often impaired.	Topical analgesics, antipyretic agents, fluids, corticosteroids, antibiotics to prevent secondary infection; hospitalization.

(continued)

Vesiculobullous Diseases (*Continued*)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Toxic epidermal necrolysis	Older adults	F	Any	Severe complement-mediated and white blood cell-mediated cytotoxic effects provoked by drugs	Severe large coalescing bullae.	IV fluids, corticosteroids, hospitalization.
Pemphigus vulgaris	46–60	2:1 (F:M)	Light-skinned persons, Jewish and Mediterranean persons	Autoimmune antibodies directed against desmoglein 3 and 1 (a component of the desmosome)	Multiple skin and mucosal bullae that rupture, hemorrhage, and crust. Lesions tend to recur in the same area, have circular or serpiginous borders, and tend to spread to adjacent areas. The Nikolsky sign is positive. Collapsed bullae are white and necrotic and produce fetid oris. Dehydration can occur if lesions are extensive.	Medical (and ophthalmologic) referral, systemic steroids, oral topical steroids, steroid-sparing and immunosuppressive agents.
Benign mucous membrane pemphigoid	>50	2:1 (F:M)	Any	Autoimmune antibodies directed against basement membrane antigens (laminin 5 and BP 180)	Bullous type produces blisters and ulcers on skin folds and inguinal and abdominal areas. Cicatricial type produces bullae and ulcers affecting the mucosa of the eyes, mouth, and genitals, which can lead to scarring. Bullae are often hemorrhagic and persist for days, then desquamate. Oral lesions that occur on the gingiva, palate, and buccal mucosa are painful and limit oral hygiene.	Topical steroids, occlusive topical steroids, dapsone, or tetracycline and niacinamide. Medical referral and systemic steroids if severe; rule out corneal involvement and internal malignancy.

(*continued*)

Ulcerative Lesions

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Traumatic ulcer	Any	M-F	Any	Traumatic insult to epithelium and underlying layers	Symptomatic, yellow-gray ulcer of variable size and shape, depending on inducing agent. Ulcers are often depressed and usually oval in shape, with erythematous border. Commonly located on labial and buccal mucosa, tongue at the borders, and hard palate. Ulcer lasts 1–2 weeks.	Palliative; remove traumatic influence.
Recurrent aphthous stomatitis	Young adult	F	Any	Instigating factor often unknown; related to defect in T-cell response; stress, trauma, and epithelial thinning	Small yellowish oval ulcer(s) with red border, located on movable nonkeratinized mucosa. Common sites include labial mucosa, buccal mucosa, floor of the mouth, tongue, and occasionally soft palate. Ulcers are tender and may be associated with a tender lymph node. Lesions develop rapidly and disappear in 10–14 days without scar formation.	Spontaneous healing in 10–14 days. If acute symptoms or recurrent and symptomatic, topical anesthetics, coagulating agents, or topical steroids may be used.
Pseudo-aphthous	25–50	F	Any	Unknown immune defect associated with folic acid, iron, or vitamin B ₁₂ deficiencies, inflammatory bowel disease, Crohn disease, gluten intolerance	Depressed yellowish round or oval ulcer located on movable nonkeratinized mucosa. Common sites include labial mucosa, buccal mucosa, floor of the mouth, tongue, and occasionally soft palate. Tongue may demonstrate atrophied papillae. Ulcers are tender, develop during deficiency state, and disappear with replacement therapy within 20 days.	Evaluate for deficiency state. If patient is deficient, then nutritional supplements (e.g., iron, vitamin B ₁₂ , folate) are recommended. Gluten abstinence may be required.

(continued)

<i>Disease</i>	<i>Age</i>	<i>Sex</i>	<i>Race/ Ethnicity</i>	<i>Cause</i>	<i>Clinical Characteristics</i>	<i>Treatment</i>
Major aphthous stomatitis	Young adult	F	Any	Instigating factor often unknown; related to defect in T-cell response	Asymmetric unilateral large ulcer with necrotic and depressed center. Ulcers have a red inflammatory border and are extremely painful. Located on soft palate, tonsillar fauces, labial mucosa, buccal mucosa, and tongue; may extend onto attached gingiva. Rapid onset. Underlying tissue is often destroyed. Lesions heal in 15–30 days with scar formation. Recurrences are common.	Spontaneous healing, sometimes with scar formation. Topical anesthetics, topical steroids, stress management; identify allergens.
Herpetiform ulceration	Late 20s	M	Any	Variant form of recurrent aphthous stomatitis; cause unknown	Multiple pinhead-sized yellowish ulcers located on movable nonkeratinized mucosa. Common sites include anterior tip of tongue, labial mucosa, and floor of the mouth. No vesicle formation. Ulcers are painful and may be associated with several tender lymph nodes. Lesions develop rapidly and disappear in 10–14 days without scar formation.	Tetracycline rinses.
Behcet syndrome	20–30	3:1 (M:F)	Asian, Mediterranean, Anglo	Delayed hypersensitivity reaction and vasculitis triggered by the presentation of human leukocyte antigens (HLA-B51) or environmental antigens, such as viruses, bacteria, chemicals, and heavy metals	Eye: conjunctivitis, iritis; Genital: ulcers; Oral: painful aphthouslike ulcers on labial and buccal mucosa; Skin: maculopapular rash and nodular eruptions. Oral ulcers are often an initial sign of the disease onset. Arthritis and gastrointestinal symptoms may be concurrent. Recurrences, exacerbations, and remissions are likely.	Topical and systemic steroids.

(continued)

Disease	Age	Sex	Race/ Ethnicity	Cause	Clinical Characteristics	Treatment
Granulomatous ulcer (tuberculosis, histoplasmosis)	Older adult	M-F	Any	Infection by <i>Mycobacterium tuberculosis</i> or <i>Histoplasmosis capsulatum</i>	Asymptomatic, cobblestoned ulcer that usually occurs on dorsum of tongue or labial commissure. Cervical lymphadenopathy and primary respiratory symptoms often are concurrent. Onset of oral disease follows lung infection lasting several weeks to months. Oral ulcer may persist for months to years if underlying disease not treated.	Biopsy, histologic examination. Tuberculosis: drug combination of isoniazid, rifampin, rifapentine, ethambutol, streptomycin, and pyrazinamide. Histoplasmosis: amphotericin B.
Squamous cell carcinoma	>50	2:1 (M:F)	Any	Mutagenesis of genes that regulate cell growth and apoptosis caused by carcinogens (tobacco, alcohol, and human papillomavirus)	Nonpainful yellowish ulcer with raised red indurated borders commonly located on posterior third of the lateral border of tongue, ventral tongue, lips, and floor of the mouth. Associated features may include numbness, leukoplakia, erythroplakia, induration, fixation, fungation, and lymphadenopathy. Carcinoma has a slow onset and is often noticed after a recent increase in size.	Surgery, radiation therapy, or chemotherapy. Smoking and alcohol cessation.
Chemotherapeutic ulcer	15–30 and older adult	M-F	Any	Inhibition of rapidly reproducing cells by chemotherapeutic drugs	Irregular ulcerations of the lips, labial and buccal mucosa, tongue, and palate. Red inflammatory border is often lacking. Hemorrhage is likely when ulcers are deeply situated. Lesions are extremely painful and usually limit mastication and swallowing. Develops during second week of chemotherapy. Secondary infection with oral microorganisms is likely.	Antimicrobial rinses to prevent secondary infection. Topical anesthetics, palliative elixirs, and intravenous fluids.

Glossary

Abdomen: The part of the body lying between the thorax (chest) and pelvis.

Abfraction: Means “to break away,” a term used in dentistry to define the loss of tooth structure at—or under—the cementoenamel junction caused by abnormal tooth flexure. The condition is controversial, and current evidence suggests that it exists only as a hypothetical component of cervical wear.

Abrasion: Wearing down or rubbing away by friction.

Acantholysis: Loss of coherence between epidermal or epithelial cells.

Acquired hypodontia: The absence of one or several teeth as a result of trauma, caries, periodontal disease, extraction, or other factors that occur after birth.

Acquired pellicle: A thin coating, derived mainly from salivary glycoproteins, which forms over the surface of a tooth crown when it is exposed to saliva.

Acute: Having severe symptoms and a short course.

Adrenal gland: A small endocrine gland located above the kidney that secretes (a) endogenous glucocorticosteroids, which control digestive metabolism; (b) mineralocorticoids, which control sodium and potassium balance; (c) sex hormones; and (d) catecholamines (epinephrine and norepinephrine), which alter blood pressure and heart function.

Adrenalectomy: Surgical removal of the adrenal gland.

Aesthetic: Pertaining to the appearance of oral or dental structures or the pleasing effect of dental restorations or procedures.

Afunctional: Not functioning or working.

Agensis: Complete absence of a structure or part of a structure caused by failure of tissue development.

AIDS: Acronym for *acquired immune deficiency syndrome*, reserved for patients infected with HIV (human immunodeficiency virus). It also refers to the terminal stage of the disease.

Allergen: A substance that induces hypersensitivity or an allergic reaction.

Amalgam: An alloy used to restore teeth, composed mainly of silver and mercury.

Amelogenesis: The formation of the enamel portion of the tooth.

Amelogenesis imperfecta: An autosomal dominant or X-linked disorder that leads to improper development of the dental enamel. The enamel can be lacking, hypoplastic, or hypocalcified. In this condition, the enamel is very thin and friable and frequently stained in various shades of brown.

Amputation: Strictly, this term refers to the removal of a limb, such as an arm, or of an appendage, such as a finger. With reference to a neuroma, however, amputation means a tumor of nerve tissue that results from severing of a nerve.

Amputation caries: Severe caries that results in complete loss of the tooth crown, often at the level of the gingiva.

Anaerobic: Lacking oxygen.

Analgesic: A drug or substance used to relieve pain.

Analogous: Having similar properties.

Anaplastic: Pertaining to adult cells that have changed irreversibly toward more primitive cell types. Such changes are often malignant.

Anergy: State of immune unresponsiveness. In allergy testing, this state occurs when the skin does not respond to an allergen.

Angioma: A tumor made up of blood or lymph vessels.

Angiogenesis: The process of developing new blood vessels.

Ankylosis: A condition marked by fusion of bones, often as a result of injury or inflammation, that results in loss of function. In dentistry, the term is used to mean when a tooth root is fused to the alveolar bone.

Anodontia: Congenital condition in which all the teeth fail to develop.

Anomaly: Deviation from normal.

Anorexia: A lack or loss of appetite for food.

Anterior: Located toward the front (opposite of *posterior*).

Antibiotic: A chemical compound that inhibits the growth or replication of certain forms of life, especially pathogenic organisms, such as bacteria or

- fungi. Antibiotics are classified as either biostatic or biocidal.
- Antibiotic sensitivity:** Testing a suspected organism to see whether it is sensitive to destruction by one or more specific antibiotics.
- Antibody:** A protein produced in the body in response to stimulation by an antigen. Antibodies react specifically to antigens in an attempt to neutralize these foreign substances.
- Antigen:** A substance, usually a protein, that is recognized as foreign by the body's immune system and stimulates formation of a specific antibody to the antigen.
- Antipyretic:** A drug or substance used to relieve fever.
- Aplasia:** Absence of an organ or organ part resulting from failure of development of the embryonic tissue of origin.
- Apposition:** Cell growth in which layers of material are deposited on already existing ones.
- Arthralgia:** Pain in one or more joints.
- Aspiration:** The withdrawal of fluid, usually into a syringe.
- Asymptomatic:** A lack of symptoms in the patient.
- Atherosclerosis:** A condition of degeneration and hardening of the walls of arteries caused by fat deposition.
- Atopy:** Hypersensitivity or allergy caused by hereditary influences.
- Atrophic:** A normally developed tissue that has decreased in size.
- Attrition:** The loss of tooth structure (wear) by mechanical forces from frequent occlusal loading from opposing teeth.
- Atypical:** Pertaining to a deviation from the normal or typical state.
- Autoimmune disorder:** A condition that occurs when the immune system mistakenly attacks and destroys healthy body tissue. To date, there are more than 80 types of autoimmune disorders.
- Autoinoculation:** To inoculate with a pathogen, such as a virus from one's own body. An example would be to spread herpesvirus from one's own mouth or lips to one's finger.
- Autosomal dominant:** One of several ways that a trait (features of a disorder) can be passed down through families. It occurs when an abnormal *gene* (from one of the 22 pair of nonsexual chromosomes) from one parent is passed down to a son or daughter such that the child inherits the disease and the gene is passed down to their children.
- Axial inclination:** The tilt the long axis of the tooth root has in the jaw in the anteroposterior plane.
- Bacterial plaque:** A collection of bacteria, growing in a deposit of material on the surface of a tooth, that can cause disease.
- Bacteriostatic:** The property of inhibiting the growth of bacteria.
- Basal cell layer:** The bottom-most layer of the epidermis composed of *dividing cells* that are anchoring the epithelium to the deeper *dermis*.
- Benign:** A tumor that does not metastasize; generally not dangerous to health, and treatment cures the condition.
- Bifid uvula:** A cleft or split (small or large) in the uvula (fleshy mass that hangs from the soft palate).
- Bilateral:** On both sides of the body or mouth.
- Biofilm:** A well-organized community of bacteria that adheres to surfaces and is embedded in an extracellular slime layer.
- Biopsy:** Excision of living tissue for the purpose of examination by a pathologist.
- Bosselated:** Covered with bumps.
- Bruxism:** A habit related to stress or a sleep disorder characterized by grinding one's teeth.
- Bulbous root:** Enlarged rounded root.
- Bulimia:** An eating disorder characterized by frequent periods of excessive food consumption followed by the purging of the ingested food by vomiting or the use of laxatives.
- Bulla:** A circumscribed, fluid-containing, elevated lesion of the skin that is >1 cm in diameter.
- Cadherin:** A family of adhesion proteins that makes up the desmosome and other structures.
- Calculus angularis:** A slightly enlarged soft tissue fold on the buccal mucosa near the corner of the mouth.
- Candida albicans:** A diploid fungus (form of yeast) that causes opportunistic oral and genital infections.
- Candidiasis:** An infection, typically of the mucosa or skin, caused *Candida albicans*.
- Carcinogen:** An agent that induces cancer.
- Carcinoma:** A malignant growth made up of epithelial cells that are capable of infiltration and metastasis. Carcinoma is a specific form of cancer.
- Cellulitis:** A spreading, diffuse, edematous, and sometimes suppurative (pus-producing) inflammation in cellular tissues.
- Cervical enamel extensions:** Spicules of enamel that project beyond the cemento-enamel junction.
- Cervical lymphadenopathy:** Abnormally large lymph nodes in the neck, often caused by lymphocyte replication in response to an allergen or infectious organism.
- Chancere:** Painless ulceration formed during the primary stage of *syphilis*.
- Chemotaxis:** Taxis or movement of cells in response to chemical stimulation.
- Chemotherapy:** Treatment by chemical substances that have a specific effect on the microorganisms causing the disease. This term is usually reserved for the treatment of cancer with the use of drugs that inhibit rapidly reproducing cells. Side effects are possible.
- Choristoma:** A lesion that contains histologically normal tissues in an ectopic (abnormal) location.

Chromogenic bacteria: Bacteria that produce various colors during growth.

Chronic: Persisting for a long time; when applied to a disease, *chronic* means that there has been little change or extremely slow progression for a long period.

Cicatricial: Pertaining to a scar.

Cicatrix: A scar; the fibrous tissue that is formed after a wound heals.

Circumscribed: Encircled; curved boundaries of a lesion.

Cirrhosis: A chronic disease of the liver characterized by degenerative changes in the liver cells, the deposition of connective tissue, and other changes. The result is that the liver cells stop functioning and the flow of blood through the liver decreases. There are many causes of cirrhosis, including infection, toxic substances, and long-term alcohol abuse.

Class I caries: Decay affecting the fissural surface of the occlusal portion of a posterior tooth.

Class II caries: Decay affecting the interproximal surface of a posterior tooth below the contact point between teeth.

Class III caries: Decay affecting the interproximal surface of an anterior tooth below the contact point between teeth.

Class IV caries: Decay affecting the interproximal surface of a tooth, including the incisal line angle.

Class V caries: Decay affecting the facial or lingual surface of a tooth, often at the gingival margin where plaque accumulates.

Clavicle: The collar bone, connecting the shoulder bone (scapula) to the chest bone (sternum).

Cleft lip: A developmental abnormality resulting in a split in the lip that is evident at the time of birth. Drug intake and smoking tobacco during pregnancy, as well as genetic factors, are contributory.

Cleft palate: A developmental abnormality resulting in a split in the palate that is evident at the time of birth. Delayed eruption and missing teeth often accompany this condition.

Cleidocranial dysplasia: A disorder involving the abnormal development of bones in the clavicle (cleido = collar bone) and skull (cranial = head).

Coagulation: The process of clotting, usually of blood. Clotting is the natural means by which bleeding ceases when a vessel has been severed.

Collagen: A protein present in the connective tissue of the body.

Coloboma: A developmental defect that may affect various parts of the eye, characterized by a missing part of the structure affected. For example, a coloboma of the lower eyelid means a missing part of the lower eyelid.

Commissural lip pits: Small, dimplelike depressions or pits found at the corner of the lips. Although rare, it

is one of the more commonly occurring congenital malformations of the lower lip. The condition may be inherited.

Commissure: The junction of the upper and lower lips at the corner of the mouth.

Complement: A series of enzymatic proteins in normal serum that, in the presence of a specific sensitizer, can destroy bacteria and other cells. C1 through C9 are the nine components of complement that combine with the antigen-antibody complex to produce lysis.

Complete cleft lip: A congenital condition involving a large split in the lip that involves the structures of the nose and often cleft palate.

Complete cleft palate: A congenital condition involving a large split in the palate that permits communication between the oral and nasal cavities, typically involving the incisive foramen.

Concrescence: A condition in which the *cementum* of two adjacent tooth roots are fused together.

Concretion: A hardened mass, such as calculus.

Concurrent: One or more conditions, events, or findings occurring at the same time.

Congenital: Present at, or existing from, the time of birth.

Congenital epulis: A proliferation of cells resulting in a soft tissue mass of the alveolar ridge, palate or tongue.

Congenital lymphangioma: A benign enlargement consisting of a mass of lymphatic vessels that are present at the time of birth.

Congenital syphilis: An infectious disease caused by a spirochete (*Treponema pallidum*) that is transmitted from mother to fetus during pregnancy. If untreated, the child is born with the infection that can damage many organs and tissues.

Connective tissue: The tissue that comprises the dermis and layers under the epithelium, characterized by collagenous, elastic, and reticular fibers; adipose tissue; blood vessels; cartilage; and bone. It forms the supporting and connecting structures of the body.

Constitutional symptoms: Symptoms affecting the whole body, such as fever, malaise, anorexia, nausea, and lethargy.

Cornified: A process whereby a tissue, usually epithelium, becomes rough and thickened in its outer coating.

Culture: The propagation of an organism in a medium conducive to growth.

Cunnilingus: Oral sex of the female genitalia.

Cusp of Carabelli: A small additional cusp on the mesiolingual surface of the maxillary *molars*.

Cyst: A pathologic epithelium-lined cavity, usually containing fluid or semisolid material.

Cytokines: A group of signaling proteins involved in inflammation and cell-to-cell communication.

Cytologic: Pertaining to the scientific study of cells.

- Cytopathic:** Pertaining to or characterized by pathologic changes in cells.
- Debilitation:** The process of becoming weakened.
- Deciduous tooth:** The primary dentition, or baby teeth. The normal number is 20.
- Deglutition:** The process of taking a substance through the mouth and throat into the esophagus. Deglutition is a stage of swallowing.
- Dehiscence:** An opening within an organ. In dentistry, the term is used to mean the loss of alveolar bone over the buccal or lingual aspect of the root, which leaves a characteristic oval root-exposed defect.
- Dehydration:** The removal of water from a substance. Prolonged fever and diarrhea cause dehydration.
- Dens evaginatus:** A developmental anomaly that appears as a small, extra, dome-shaped, cusplike structure that emanates from the occlusal surface of a posterior tooth.
- Dens in dente:** A developmental anomaly that results from enamel and dentin folding inwardly toward the pulp. There are coronal and radicular forms.
- Dental caries:** A progressive destruction of the mineralized structures (enamel and dentin) of the teeth resulting from bacterial infection. The bacteria are found in plaque and use fermentable carbohydrates for food with the by-product being acid formation.
- Dental lamina:** The embryonic tissue of origin of the teeth.
- Dental lamina cysts:** Cysts that develop from the embryonic tissue that form teeth. Normally, the dental lamina disintegrates into small clusters of epithelium and is resorbed, but in this case, it becomes cystic.
- Dentin dysplasia:** A genetic (autosomal dominant) disorder involving abnormal dentin development of the primary and permanent teeth. Two types are described: Type I is the radicular type, and type II is the coronal type. In the radicular type, the roots of teeth are short, the *pulp* chambers are constricted, and spontaneous periapical radiolucencies may be evident. In the coronal type, the pulps have a “thistle tube” appearance.
- Dentinoenamel junction (DEJ):** The line or junction between the dentin and enamel.
- Dermis:** The connective tissue layer below the epidermis (skin) that contains nerve endings, sweat and sebaceous glands, blood vessels, and lymph vessels.
- Desmosome:** An intercellular bridge between epithelial cells. The bridge is made of proteins and tonofilaments.
- Developmental:** Pertaining to growth to full size or maturity.
- Diascopy:** The examination of tissue under pressure through a transparent medium. For example, suspected vascular lesions are examined by pressing a glass slide over an abnormality to see whether the reddish tissue turns white. Because blood flows through vascular lesions, pressure causes them to turn white and thus helps to confirm the diagnosis.
- Dilaceration:** A bent root, usually affecting the apical half or third of the root.
- Distal:** Farthest from a point of reference. In dentistry, *distal* describes the surface farthest from the midline of the patient.
- Distal drift:** A tooth that moves distally often because of available space that results from a missing adjacent tooth.
- Dorsal:** Directed toward or situated on the back surface (opposite of *ventral*).
- Ducts of Ravini:** Ducts that lie beneath the tongue and permit saliva to enter the mouth from the sublingual glands.
- Dysphagia:** Difficulty swallowing.
- Dysphonia:** Difficulty speaking.
- Dysplasia:** An abnormality of development and maturation characterized by the loss of normal cellular architecture.
- Dysplastic:** Pertaining to an abnormality of development. This term is often used to describe the appearance of abnormal, premalignant cells under the microscope. The cells begin to lose their normal maturation pattern and have abnormally shaped, hyperchromatic nuclei.
- Dyspnea:** Labored or difficult breathing.
- Ecchymoses:** Large reddish-blue areas caused by the escape of blood into the tissues, commonly referred to as a bruise. Ecchymoses do not blanch on diascopy.
- Ecosystem:** The interaction of living organisms and nonliving elements in a defined area.
- Ectodermal:** Pertaining to the outermost of the three primitive germ layers of an embryo. The middle layer is the mesoderm, and the innermost layer is the endoderm. Ectodermal structures include the skin, hair, nails, oral mucous membrane, and the enamel of the teeth.
- Ectodermal dysplasia:** A hereditary condition characterized by abnormal development of the skin, hair, nails, teeth, and sweat glands.
- Ectopic:** Located in an abnormal place. The ectopic tissue or structure may or may not be normal.
- Ectopic eruption:** Abnormal eruption location of a tooth.
- Edema:** Abnormal amounts of fluid in the intercellular spaces, resulting in visible swelling.
- Edematous:** An excessive accumulation of serous fluid in tissue spaces or a body cavity that produces swollen soft tissues.
- Edentulism:** A condition without teeth. The complete loss of all natural teeth.
- Emanate:** To give off or flow away from.
- Embryonic:** Pertaining to the earliest stage of development of an organism.

- Enamel hypoplasia:** A disturbance in the development of enamel marked by deficient or defective enamel matrix formation, resulting in thin and hypocalcified enamel.
- Enamel pearls:** A concretion of enamel that is often dome shaped in an abnormal location. Pearls can appear at the cemento-enamel junction or root surface and contribute to periodontal disease; rarely, they appear within dentin.
- Encephalitis:** Inflammation of the brain.
- Endocrinopathy:** A disease or abnormal state of an endocrine gland.
- Endodermal:** Pertaining to the innermost of the three primitive germ layers of an embryo. Endodermal structures include the epithelium of the pharynx, respiratory tract (except the nose), and the digestive tract.
- Epidermis:** The outermost layer of skin made of stratified squamous epithelium that protects the body surface.
- Epistaxis:** Bleeding from the nose.
- Epithelium:** The cellular makeup of skin and mucous membranes.
- Epitope:** The antigenic target of antibodies (IgG, IgM, IgA).
- Epulis:** A generic term used to describe any nodular or tumorous enlargement of the gingiva or alveolar mucosa.
- Erosion:** The wearing away of teeth through the action of chemical substances, or a denudation of epithelium above the basal cell layer.
- Eruption:** An emergence from beneath a surface. For teeth, *eruption* means growth into the oral cavity; it may also refer to the development of skin lesions.
- Eruption cyst:** A fluctuant, fluid cyst that can appear 2 to 3 weeks before the eruption of a tooth. They are most often bluish, soft, associated with primary tooth eruption, and disappear upon tooth eruption.
- Erythema:** Redness in a tissue area.
- Erythematous:** Characterized by a redness of the tissue because of engorgement of the capillaries in the region. Erythematous lesions blanch on diascopy.
- Erythroplastic:** Characterized by a reddish appearance. This term implies abnormal tissue proliferation in the reddish area.
- Eschar:** A slough of epithelium often caused by disease, trauma, or chemical burn.
- Esophagus:** The flexible muscular tube that connects the mouth with the stomach.
- Everted:** Folded or turned outward.
- Exacerbation:** An increase in severity.
- Exanthematic:** Characterized by the development of an eruption or rash.
- Excisional biopsy:** To completely remove a mass of tissue for the purpose of scientific (histologic) analysis.
- Exophytic:** An outwardly growing lesion.
- Exostosis:** A benign bony outgrowth on the surface of bone.
- Extensor surface:** Because the arms and legs can be extended or tensed by the appropriate extensor or tensor muscles, the anterior surface is referred to as the extensor surface, and the posterior surface is referred to as the tensor surface.
- Extirpate:** To completely remove or eradicate.
- Extremity:** A limb of the body, such as an arm or leg.
- Extrinsic stains:** Stains that come from external sources.
- Exudate:** Material that has escaped from blood vessels into tissue or onto the surface of a tissue, usually because of inflammation.
- Factitial:** Self-induced, as in factitial injury.
- Fascial plane:** Spaces between adjacent bundles of fascia that cover muscles. Infection often spreads along these planes.
- Fellatio:** Oral stimulation of the penis.
- Fenestration:** A perforation or opening in a tissue.
- Fetor oris:** An unpleasant or abnormal odor emanating from the oral cavity.
- Fibroma:** A benign tumor of fibrous (collagen) tissue.
- Field cancerization:** Malignant growths occurring in multiple sites of the oral cavity. The oral tissues have often been exposed to a carcinogen for a long time.
- Fissural caries:** Dental decay of the occlusal surface of a posterior tooth along enamel fissures and grooves.
- Fissure:** A narrow slit or cleft.
- Fluctuant:** Strictly, this term describes a palpated, wavelike motion that is felt in a fluid-containing lesion. In this text, the term is frequently used to describe a soft, readily yielding mass on palpation.
- Fluorosis:** An abnormal condition caused by excess intake of fluorine or fluoride that is characterized by mottling and discoloration of the teeth.
- Focal:** A specific location or focus.
- Fontanelle:** One of several soft spots on the skull of infants and children in which the bones of the skull have not yet completely united. In these areas, the brain is covered only by a membrane beneath the skin.
- Fovea palatinae:** Minor salivary gland openings close to the junction of the soft and hard palate.
- Frenum:** A fold of mucous membrane that limits the movement of an organ or organ part. For example, the lingual frenum limits tongue movement, and the labial frenula limit lip movements.
- Frontal bone:** The skull bone that forms the forehead. The frontal bone contains an air space called the frontal sinus.
- Furcal:** Pertaining to or associated with the part of a multirrooted tooth where the roots join the crown.
- Fusion:** An abnormality caused by the joining of two teeth during embryologic development resulting in one large tooth.

- Ganglion:** A collection of cell bodies of neurons outside of the central nervous system. A ganglion is essentially a terminal through which many peripheral circuits connect with the central nervous system.
- Gardner syndrome:** An inherited autosomal dominant disease that has the features of osteomas and intestinal polyps. The polyps have a propensity to become malignant during early adulthood.
- Gastroenterologist:** A medical specialist whose field is disorders of the stomach and intestines.
- Gastrointestinal:** Pertaining to the stomach and intestine.
- Gemination:** To arrange or occur in pairs. A tooth that splits during embryologic development into two teeth that remain connected.
- Genetic counseling:** A form of patient counseling in which the transmission of inherited traits is discussed.
- Genial tubercles:** Bony spines located on the lingual aspect of the mandible at the midline below the incisor roots that serve as the attachment of the geniohyoid and genioglossus muscles.
- Genodermatosis:** A hereditary skin disease.
- Gingival crevicular fluid:** Serum fluid emitted from the periodontal pocket. It contains inflammatory proteins (cytokines). The amount of fluid increases during periodontal disease.
- Gingivectomy:** Surgical removal of gingival tissue.
- Gingivitis:** Inflammation of the gums, typically because of the accumulation of dental plaque.
- Glaucoma:** A disease of the eye characterized by increased intraocular pressure. This condition is often asymptomatic and, if not recognized or treated, leads to blindness.
- Glossal:** Pertaining to or associated with the tongue.
- Glossodynia:** Burning sensation of the tongue.
- Glucan:** A sticky substance (polysaccharide) secreted by bacteria in dental plaque that is composed of chains of D-glucose monomers linked by glycosidic bonds.
- Glucose:** A form of sugar that is the most important carbohydrate in the body's metabolism.
- Glucosuria:** The presence of an abnormal quantity of glucose in the urine. A sign of diabetes mellitus.
- Granuloma:** A collection of epithelioid macrophages surrounded by a ring of lymphocytes. They can form small nodules and are an outcome of a necrotic pulp.
- Granulomatous:** Pertaining to a well-defined area that has developed as a reaction to the presence of living organisms or a foreign body. The tissue consists primarily of histiocytes.
- Gravid:** Pregnant.
- Gutta percha points:** An inelastic natural latex produced from the sap of trees that is shaped into cylindrical points that are used in endodontics to fill a cleaned and shaped root canal.
- Halitosis:** An unpleasant odor of the breath or expired air.
- Hamartoma:** A tumorlike nodule consisting of a mixture of normal tissue usually present in an organ but existing in an unusual arrangement or an unusual site.
- Hapten:** An incomplete allergen. When combined with another substance to form a molecule, a hapten may stimulate a hypersensitivity or allergic reaction.
- Hematoma:** A large ecchymosis or bruise caused by the escape of blood into the tissues. Hematomas are blue on the skin and red on the mucous membranes. As hematomas resolve, they may turn brown, green, or yellow.
- Hematopoietic:** Pertaining to the production of blood or of its constituent elements. Hematopoiesis is the main function of the bone marrow.
- Hematuria:** The presence of blood in the urine.
- Hemidesmosome:** An intercellular bridge between epithelial cells and the basement membrane.
- Hemihypertrophy:** The presence of hypertrophy on one side only of a tissue or organ. In facial hemihypertrophy, for example, one half of the face is visibly larger than the other.
- Hemoglobin:** The iron-containing pigment of the erythrocytes. Its function is to carry oxygen to the tissues. One of the causes of anemia is a deficiency of iron, causing patients to look pale and feel tired.
- Hemolysis:** Generally speaking, this term refers to the disintegration of elements in the blood. A common form of hemolysis occurs during anemia and involves lysis or the dissolution of erythrocytes.
- Hemorrhage:** Bleeding; the escape of blood from a severed blood vessel.
- Hemostasis:** The stoppage of blood flow. This can occur naturally by clotting or artificially by the application of pressure or the placement of sutures.
- Hepatosplenomegaly:** The simultaneous enlargement of the liver and spleen.
- Hereditary:** Transmitted or transmissible from parent to offspring; determined genetically.
- Herpes simplex virus (HSV):** A transmissible DNA virus belonging to the human herpesvirus family that causes gingivostomatitis, mucosal ulcers, and perioral disease. This virus establishes latency in neurons and reactivates to cause recurrent ulcers (fever blisters).
- Hiatal hernia:** Protrusion of any structure through the hiatus of the diaphragm. Affected patients are prone to indigestion.
- Histiocyte:** A large phagocytic cell from the reticuloendothelial system. The reticuloendothelial system is a network made up of all of the phagocytic cells in the body, which include macrophages, Kupffer cells in the liver, and the microglia of the brain.
- Histology:** The microscopic study of the structure and form of the various tissues making up a living organism.

Histoplasmosis: A disease caused by the fungus *Histoplasma capsulatum* that primarily affects the lungs, but also affects the eyes and infrequently the tongue.

Human papillomavirus (HPV): A small transmissible DNA virus that causes human disease. More than 100 different HPVs have been identified. The “low-risk” types cause benign growths, such as warts and papillomas; the “high-risk” types can cause cancer.

Hutchinson triad: A pattern of presentation for congenital syphilis that consists of abnormal teeth (Hutchinson incisors and mulberry molars), interstitial keratosis, and deafness as a result of damage to the eighth cranial nerve.

Hypercementosis: A condition characterized by increased cementum deposition on the roots of teeth. Any part of the root may be affected; however, the apical two thirds is most common. Local or systemic factors may cause this condition.

Hyperdontia: A condition or circumstance characterized by one or more extra (or supernumerary) teeth.

Hyperemia: The presence of excess blood in a tissue area.

Hyperglycemia: The presence of excessive sugar or glucose in the bloodstream.

Hypermenorrhea: Excessive uterine bleeding of unusually long duration at regular intervals.

Hyperorthokeratosis: Keratin is the outermost layer of epithelium as seen under the microscope and is seen in two forms: orthokeratin and parakeratin. Orthokeratin has no visible nuclei within the outer layer, whereas nuclei are present in parakeratin. Hyperorthokeratosis is the presence of excess orthokeratin.

Hyperplasia: An increase in the size of a tissue or organ caused by an increase in the number of constituent cells.

Hyperplastic: Pertaining to hyperplasia; tissue that displays hyperplasia features.

Hypersensitivity: Generally, this term means an abnormal sensitivity to a stimulus of any kind; however, it is often used with specific reference to some form of allergic response.

Hypertension: High blood pressure.

Hypertrophy: An increase in the size of a tissue or organ caused by an increase in the size of constituent cells.

Hypocalcification: Less than normal amount of calcification.

Hypodontia: The congenital absence of one or several teeth as a result of agenesis.

Hypoplasia: Incomplete development of a tissue or organ; a tissue reduced in size because of a decreased number of constituent cells.

Hypopyon: Pus in the anterior chamber of the eye.

Hypotension: Low blood pressure.

Hypotrichosis: A condition of lack of hair growth.

Iatrogenic: A condition or disorder resulting from a complication of medical treatment.

Idiopathic: Arising from an obscure or unknown cause.

Ileum: The distal or terminal portion of the small intestine, ending at the cecum, which is a blind pouch forming the proximal or first part of the large intestine.

Ilium: The lateral or flaring part of the pelvic bone, otherwise known as the hip.

Immunofluorescence: A laboratory assay used by pathologists and researchers that applies known antibodies to tissue to identify the presence and location of specific antigens or proteins.

Incipient lesion: A beginning lesion; in an initial stage.

Incisional biopsy: The removal of a portion of suspected abnormal tissue for microscopic study.

Incisive canal: The bony canal located within the palatal bone that supports the nasopalatine nerve and blood vessels and exits at the incisive foramen behind the maxillary central incisors.

Incisive papilla: A slightly elevated papule of normal tissue on the palate in the midline immediately posterior to the central incisors. Immediately beneath this structure lies the incisive canal.

Induration: Characterized by being hard; an abnormally hard portion of a tissue with respect to the surrounding similar tissue, often used to describe the feel of locally invasive malignant tissue on palpation.

Infant: A human baby from birth to 2 years of age.

Infarct: A localized area of ischemic necrosis resulting from a blockage of the arterial supply or the venous drainage of tissue. Ischemic necrosis is dead tissue resulting from an inadequate blood supply. An example is a heart attack, which is an infarct of heart muscle.

Inflammation: A bodily process involving white blood cells and chemicals that results from infection, irritation, or injury and produces tissue redness, warmth, swelling, and pain.

Insulin: A protein hormone secreted by the islets of Langerhans of the pancreas that helps move glucose from the blood to tissues. Insulin deficiency produces hyperglycemia, otherwise known as diabetes mellitus.

Integrins: A large group of heterodimeric transmembrane proteins that serve as receptors for extracellular matrix and cell surface proteins.

Intrinsic staining: A term used in dentistry to indicate discoloration from staining that occurs from within the tooth.

Invaginate: To fold and grow within, in the manner of a pouch.

Iris: The part of the eye that is blue, gray, green, or brown. It is a muscular tissue, and its function is to constrict and dilate the pupil. The pupil is the black-appearing portion in the middle of the iris that allows light into the eye.

- Iritis:** Inflammation of the iris that is often caused by viral infection or rheumatoid disease. The main symptom of iritis is photophobia (aversion to light).
- Irreversible pulpitis:** Inflammation of the pulp that can not be reversed that eventually results in death of the pulp. Pain is a key feature of this condition.
- Ischemia:** A deficiency of blood to a body part, usually caused by constriction or blockage of a blood vessel.
- Kaposi sarcoma:** A malignant tumor of vascular tissue. Once rare in the Americas, it is now seen frequently in patients with AIDS. The lesions are red-purple in appearance and may be seen anywhere on the skin, especially on the face and in the oral cavity.
- Keratin:** A strong protein and major component of skin, hair, nails, and teeth.
- Keratinization:** The formation of microscopic fibrils of keratin in the keratinocytes (keratin-forming cells). In the oral cavity, the term is used to describe changes in the outer layer of the epithelium.
- Keratotic:** A condition of the skin characterized by the presence of horny growths. On the oral mucous membrane, keratotic tissue usually looks white; the term implies a thickening of the outer layer of the oral epithelium.
- Kilodalton:** A unit of mass being one thousand times that of a dalton. A dalton is one sixteenth the mass of an oxygen atom.
- Lamina dura:** The thin compact bone that surrounds the socket (alveolus) of a tooth.
- Lamina propria:** The layer of connective tissue immediately beneath the epithelium of the oral mucosa.
- Laryngeal:** Pertaining to the larynx, which is a part of the airway. It is located between the pharynx at the back of the oral cavity and the trachea at the beginning of the lungs. The larynx contains the vocal cords, which make audible sounds.
- Lateral:** Pertaining to or situated at the side.
- Lateral vaults:** The region of the palate adjacent to the posterior teeth.
- Leptomeninges:** The two more-delicate components of the meninges: the pia mater and the arachnoid.
- Lesion:** A site of structural or functional change in body tissues that is produced by disease or injury.
- Leukoplakia:** A white patch that cannot be rubbed off and that does not clinically represent any other condition.
- Lipid:** Fat or fatty; a naturally occurring substance made up of fatty acids.
- Lipoma:** A benign soft tissue tumor composed of fat cells (adipocytes).
- Lobulated:** Made up of lobules, which are smaller divisions of lobes. Many structures are divided into lobes and lobules, such as the brain, lung, and salivary glands. Some pathologic lesions are described as lobulated when the lesion is divided into smaller parts.
- Lymphadenitis:** Inflammation of lymph nodes generally resulting in enlargement and tenderness.
- Lymphadenopathy:** Enlarged lymph nodes that may or may not be tender. The enlargement results from increased number of lymphocytes presenting in the node.
- Lymphoblastic:** Pertaining to a cell of the lymphocytic series; the term implies proliferation. Lymphoblastic is one of the forms of leukemic cancer of the leukocytes characterized by the presence of malignant lymphoblasts or immature lymphocytes.
- Lymphocyte:** A variety of leukocyte that is important to the immune response and that arises in the lymph nodes. Lymphocytes can be large or small; they are round and nongranular and are classified as either T or B lymphocytes.
- Macrocheilia:** Abnormally large lips.
- Macrodontia:** Teeth that are considerably larger than normal.
- Macule:** A spot or stain on the skin or mucous membrane that is neither raised nor depressed. Some examples of macules include café au lait spots, hyperemia, erythema, petechiae, ecchymoses, purpura, oral melanotic macules, and many others illustrated in this atlas.
- Malaise:** A constitutional symptom that describes a feeling of uneasiness, discomfort, or indisposition.
- Malignant:** A neoplastic growth that is not usually encapsulated, grows rapidly, and can readily metastasize.
- Marrow space:** The space in the trabecular bone composed of red or yellow marrow. Red marrow is where hematopoiesis occurs. Yellow marrow is composed mostly of fat. The size of the marrow space can indicate pathologic processes (e.g., anemia, thalassemia, trauma, infection, or tumors).
- Mastication:** Chewing.
- Medial:** Situated toward the midline (opposite of lateral).
- Median palatal raphe:** A fibrous band of soft tissue that is found in the midline of the palate.
- Melena:** Darkened or black feces that are caused by the presence of blood pigments; a sign of intestinal bleeding.
- Meningitis:** Inflammation of the meninges, which are the three membranes covering the brain and spinal cord (the dura mater, arachnoid, and pia mater). Meningitis produces both motor and mental signs, such as difficulty in walking and confusion.
- Mesenchymal:** The meshwork of embryonic connective tissue in the mesoderm that gives rise to the connective tissue of the body, blood vessels, and lymph vessels.
- Mesial:** Toward the front, anterior, or midline. The mesial surface of teeth is the side of the tooth closest to the midline. The five surfaces of teeth are mesial,

distal, occlusal or incisal, labial or facial, and lingual or palatal.

Mesiodens: A midline supernumerary tooth commonly seen in the maxillary arch.

Metastasis: To spread or travel from one part of the body to another; a term usually reserved to describe the spread of malignant tumors.

Microdontia: Teeth that are considerably smaller than normal.

Micrognathia: Jaws that are small with respect to normal.

Migration: Movement from one location to another.

Mineralized: Characterized by the deposition of mineral, often calcium and other organic salts, in a tissue. The term *calcified* is used when the mineral content is known to be calcium, whereas the term *mineralized* is more general and does not specify the exact nature of the mineral.

Monocytic leukemia: Leukemia is cancer of the leukocytes; in this condition, the predominating leukocytes are monocytes.

Morphology: Descriptive of shape, form, or structure, or the science thereof.

Mucinous saliva: Saliva that contains mucin, a large glycosylated protein produced by salivary glands (primarily the submandibular gland).

Mucopurulent: Consisting of both mucus and pus.

Mucosa: The moist epithelial lining of endodermal structures, such as the mouth, eyes, gastrointestinal tract, lungs, and genitalia.

Mutagenesis: The induction of genetic mutation.

Myelogenous leukemia: Leukemia is cancer of the leukocytes; in this instance, the predominating leukocytes are myeloid or granular (polymorphonuclear leukocytes).

Nasopharyngitis: Inflammation of the nasopharynx (the back of the nasal complex and upper throat). Sore throat, postnasal drip, and fever are common signs.

Natal teeth: Teeth that are present at birth.

Necrosis: The death of a cell as a result of injury or disease.

Neocapillary: New growth of capillaries, which are the smallest blood vessels and connect small arterioles to small venules.

Neoplasia: Characterized by the presence of new and uncontrolled cellular growth.

Neoplasm: A mass of newly formed tissue; a tumor.

Neoplastic: Tissue that has features of neoplasia.

Neural crest cells: Cells that originate from the neural crest (a transient ectodermal tissue found between the neural tube and the epidermis during embryologic development). Neural crest cells migrate from this region and differentiate into a wide variety of cell types throughout the body, giving rise to sensory and sympathetic neurons, glia cells, melanocytes, Schwann

cells, adrenomedullary cells, and components of the enteric nervous system.

Neurogenic: Originating in or from nerve tissue.

Neuroma: A tumor of nervous tissue that is usually benign and associated with numbness and pain.

Neuropathy: Any abnormality of nerve tissue.

Neutrophil: A medium-sized leukocyte with a nucleus consisting of three to five lobes and a cytoplasm containing small granules; one group of leukocytes is called *granulocytes*, and the others are *eosinophils* and *basophils*. Neutrophils make up about 65% of the leukocytes in normal blood. Also known as polymorphonuclear leukocyte, PMN, or “poly.”

Neutrophil chemotaxis: Taxis or movement of neutrophils in response to chemical substances or agents.

Nevoid basal cell carcinoma syndrome (Gorlin-Goltz syndrome): An autosomal dominant syndrome caused by a mutation in a gene known as patched (PTCH), a tumor suppressor gene located on chromosome 9q. This syndrome is characterized by multiple jaw odontogenic cysts, basal cell nevi on the skin, skeletal anomalies (bifid and other rib anomalies), and soft tissue anomalies like prominent finger pads and palmar pitting of the hands.

Nevus: A small tumor of the skin containing aggregations or theques of nevus cells; a mole. It may be flat or elevated and pigmented or nonpigmented; it may or may not contain hair.

Nodule: A circumscribed, usually solid lesion having the dimension of depth. Nodules are <1 cm in diameter.

Noncaseating: A tissue-degenerative process that forms a dry, shapeless mass resembling cheese.

Noncornified: The lower layers of epithelium (intermediate, parabasal, and basilar cells) that do not have a keratinized surface.

Nonvital tooth: A tooth with dead pulp tissue that is unable to transmit nerve signals effectively.

Nursing bottle caries: Dental decay that affects the primary teeth as a result of prolonged use of the nursing bottle that contains milk, juice, or soda pop. The maxillary anterior teeth are most commonly and can be severely affected.

Occipital bone: One of the bones that make up the skull; a thick bone at the back of the head.

Odontogenic epithelium: Epithelium that develops into teeth. After tooth development, this epithelium can remain within the jaws and become cystic.

Oligodontia: Few teeth; the presence of fewer than the normal number of teeth.

Oncogenic: Capable of causing tumor formation.

Open contact: A term that describes the interproximal contact between teeth that normally touch each other. In this instance, the interproximal space between the teeth do not contact.

Operculum: The gingival flap of tissue surrounding the crown of a partially erupted tooth.

- Opportunistic microorganism:** Microorganisms that usually are not pathogenic but become so under certain circumstances, such as an environment altered by the action of antibiotics or long-term steroid therapy. Opportunistic microorganisms cause opportunistic infections.
- Organism:** Any viable life form, such as animals, plants, and microorganisms, including bacteria, fungi, and viruses.
- Orogenital:** Refers to mouth contact with the genitalia.
- Oropharynx:** The area of the throat that is at the back of the mouth.
- Otorhinolaryngologist:** An ear, nose, and throat specialist.
- Orthodontic tooth movement:** The repositioning of teeth using removable or fixed appliances, such as braces, to straighten the alignment of teeth and improve the appearance and function of the teeth.
- Osteogenesis imperfecta:** A genetic disorder characterized by a defect in collagen that results in bones that break easily and defective dentin in teeth.
- Osteoma:** A benign tumor of bone.
- Overhang:** Defect in a restoration that extends beyond the interproximal surface of the tooth.
- Palato-gingival groove:** A defect in cementum formation that results in a linear groove on the lingual surface of a tooth root, most commonly a maxillary incisor.
- Palliative:** Treatment to relieve symptoms; not the cause of a condition.
- Pallor:** Paleness of the skin or mucous membrane; an absence of a healthy color. This sign often accompanies constitutional symptoms and anemia.
- Palpate:** To feel with the fingers or hand.
- Papilloma:** A benign pebbly growth caused by infection with human papillomavirus.
- Papillomatous:** Pertaining to a benign pebbly growth.
- Papule:** A small mass, without the dimension of depth, that is <1 cm in diameter. When described as pedunculated, a papule is on a stalk; when described as sessile, a papule is attached at its base and does not have a stalk.
- Papulonodular:** A type of lesion elevated above the surface of the skin that has features both of a papule and nodule.
- Parakeratin:** A type of epithelium consisting of the outermost layer (stratum corneum) that demonstrates keratin and small residual nuclei.
- Parakeratotic epithelium:** Incomplete keratinization characterized by retention of nuclei of cells at the uppermost level of the epithelium (stratum corneum).
- Paramedian lip pits:** Small bilateral depressions in the mucosa of the lower lip adjacent to the midline. This condition is often inherited as an autosomal dominant trait associated with van der Woude syndrome.
- Paramolar:** A supernumerary or “fourth” molar positioned distal to the third molar.
- Parietal bone:** One of the bones that make up the skull; there is one parietal bone on each side of the skull, forming the skull’s top and upper sides.
- Partial eruption:** A condition in which a tooth erupts through the gum tissue but not fully into occlusion.
- Parturition:** The delivery of the fetus from the mother; to give birth.
- Parulis:** A gumboil; a subperiosteal abscess arising from dental structures that emanates onto the gingival or alveolar mucosa.
- Patch:** Similar to a macule but larger; a large stain or spot, usually neither raised nor depressed, which may be textured.
- Patent:** The condition of being open; this term is often applied to ducts, vessels, and passages to indicate that they are not blocked.
- Pathognomonic:** Uniquely distinctive of a specific disease or condition; usually consists of signs or findings that, when present and recognized, enable the diagnosis to be made.
- Pathologic:** Pertaining to or caused by disease.
- Pathosis:** An abnormal state or condition.
- Pedunculated:** A tissue mass originating by a stalk from its base.
- Periapical:** Pertaining to or located at the apex (root end) of a tooth.
- Periapical abscess:** An abscess located at the apex (root end) of a tooth, generally as a result of infection.
- Periapical inflammation (apical periodontitis):** Inflammation located at the apex (root end) of a tooth.
- Pericoronitis:** Inflammation of the tissue overlying a partially erupted tooth.
- Perifurcal:** Pertaining to or located at the furca of a tooth, below the cemento-enamel junction where the roots fuse together.
- Perilabial:** Pertaining to the region around or near the lips.
- Perinatal:** Pertaining to the time frame around childbirth.
- Perineum:** The lower surface of the trunk; when a patient is lying down with legs spread apart, the perineum is the area from the base of the spine to the anal region to the genital area and, finally, to the crest of the mons pubis.
- Periodontal abscess:** Abscess of the gingival or periodontal tissue secondary to periodontal infection.
- Periodontitis:** Inflammation resulting from microbial infection of the structures that support the teeth.
- Perioral:** In the proximity of or around the oral cavity.
- Periorbital:** In the proximity of or around the orbit, which is the bony socket of the eye.
- Periosteum:** The thin layer of dense, irregular, connective tissue membrane that covers the outer surface of bone, excluding the joints.

Peripheral: Pertaining to the outer part, such as the edge or margin.

Permanent dentition: Succedaneous (adult) teeth, which follow the primary teeth. As there are no replacements for the permanent teeth, they should be properly taken care of if they are to last a lifetime. There are 32 permanent teeth.

Petechiae: Little red spots, ranging in size from pinpoint to several millimeters in diameter. Petechiae consist of extravasated capillary blood.

Pharyngitis: Inflammation of the pharynx, which is often painful.

Physiologic: Refers to normal body function (opposite of *pathologic*).

Pilocarpine: A drug used to stimulate salivary flow or to produce constriction of the pupil of the eye.

Pink tooth of mummery: A pink, discolored nonvital tooth that obtains its color from absorption of blood products into the dentin.

Plaque: An area with a flat surface and raised edges.

Platelet: One of the elements found in circulating blood. A platelet has a circular or disklike shape and is small; hence the term *platelet*. Platelets aid in blood coagulation and clot retraction.

Plica fimbriata: Slight folds of mucous membrane tissue located bilaterally on the ventral surface of the tongue.

Polydipsia: Excessive thirst. A sign of disease.

Polypoid: A polyplike protruding growth with a base that is equal in diameter to the surface of the mucosal lesion.

Polyuria: Excessive amounts of urine. A sign of disease.

Posterior: Directed toward or situated at the back (opposite of *anterior*).

Primary tooth: Deciduous (baby) tooth; there are 20 primary teeth.

Prognathism: A developmental deformity of the mandible that causes it to protrude abnormally.

Protostylid: An extra cusp found on the buccal surface of a molar.

Pruritus: Itching.

Pseudohyphae: Long, filamentous forms that can be seen under the microscope when *Candida albicans*, a fungal microorganism, assumes its pathogenic form.

Pulp polyp: An enlargement of pulp tissue beyond a broken down tooth crown in response to bacterial infection and inflammation. The condition is most common in primary or permanent molars in children.

Pulse: A patient's heartbeat, as felt through palpation of a blood vessel.

Punctate: Spotted; characterized by small points or punctures.

Purpuric: Pertaining to purpura, which are large bruises consisting of blood extravasated into the tissues. Bruises are bluish-purple in color.

Purulent: Containing pus.

Pustule: A well-circumscribed, pus-containing lesion, usually <1 cm in diameter.

Pyogenic granuloma: A mass of vascular granulation tissue produced in response to minor trauma or chronic irritation.

Qualitative: Of or pertaining to quality; descriptive information about what something looks and feels like.

Quantitative: Of or pertaining to quantity; descriptive information about how much of something there is or how big something is.

Radiation: In dentistry, electromagnetic energy or x-rays transmitted through space. Radiation also means divergence from a common center; one of the properties of x-rays is that, like a beam of light, they diverge from their source.

Radiolucent: A term to describe anatomic structures that allow the passage of x-rays or other radiation to a film or sensor. Radiolucent structures appear dark on the film.

Radiopaque: A term to describe anatomic structures that do not allow the passage of x-rays or other radiation to a film or sensor. Radiopaque structures appear light on the film.

Radiotherapy: Radiation therapy; the use of radiation from various sources to treat or cure malignant disorders.

Rampant caries: A rapidly progressing form of decay affecting the teeth.

Recessive inheritance: A mode of passing a gene carried by both parents that does not cause any clinical manifestation until passed to their offspring.

Recrudescence: Recurrence of signs and symptoms of a disease after temporary abatement.

Recurrent caries: Decay affecting the teeth adjacent to surfaces where decay previously occurred and a restoration was placed. Caries adjacent to a restoration margin.

Reduced enamel epithelium: The epithelium (sulcular epithelium) that remains in the periodontal tissue around a tooth crown after enamel formation is complete. Cystic degeneration of this tissue results in a dentigerous cyst.

Refractory: Not readily responsive to treatment.

Regional odontodysplasia: A rare developmental abnormality of teeth resulting in defective (thinned) enamel and dentin. On radiographs, the teeth have reduced radiodensity, producing a ghostlike appearance.

Remission: Improvement or abatement of the symptoms of a disease; the period during which symptoms abate.

Renal failure: Inability of the kidneys to function properly. A patient whose kidneys completely fail will die without renal dialysis or a kidney transplantation. One of the causes of kidney failure is prolonged hypertension (high blood pressure).

- Retinopathy:** A disease or abnormality of the retina of the eye. The retina cannot be seen without special instruments and is the part of the eye that receives and transmits visual information coming in from the pupil and lens onto the brain via the optic nerve.
- Retrognathia:** A retruded mandible.
- Reversible pulpitis:** Inflammation of the pulp that causes pain but is reversible if the cause of the inflammation is removed.
- Root caries:** Decay affecting the root surface of the tooth usually at the gingival margin. This condition only occurs when root surfaces are exposed, usually in association with gingival recession.
- Rotated tooth:** An erupted tooth that is positionally rotated from that of normal.
- Sarcoma:** A malignant growth of cells of embryonic connective tissue origin. This condition is highly capable of infiltration and metastasis.
- Sarcomatous:** Pertaining to sarcoma, which is a malignant tumor of mesenchymal tissue origin.
- Scar:** A mark or cicatrix remaining after the healing of a wound or other morbid process.
- Sclera:** The strong outer tunic of the eye, or whites of the eyes. When the sclera turns blue or yellow, it is a sign of systemic abnormality.
- Sebaceous:** Pertaining to glands that produce sebum that are often associated with hair follicles.
- Sepsis:** A morbid state resulting from the presence of pathogenic microorganisms, usually in the bloodstream.
- Septicemia:** The presence of pathogenic bacteria in the blood.
- Sequestration:** Abnormal separation of a part from the whole, such as when a piece of bone sequesters from the mandible because of osteomyelitis; the act of isolating a patient.
- Serosal:** Pertaining to the lining of internal organs, such as the intestines.
- Serpiginous:** Characterized by a wavy or undulating margin.
- Serum:** The watery fluid remaining after coagulation of the blood. If clotted blood is left long enough, the clot shrinks and the fibrinogen is depleted; the remaining fluid is the serum.
- Sessile:** Attached to a surface on a broad base; does not have a stalk.
- Shell teeth:** Teeth lacking normal dentin, producing a shell appearance, which are often associated with dentinogenesis imperfecta.
- Shovel-shaped incisors:** A syndrome seen in Native Americans, Canadians, Eskimos, and Hispanics that is associated with prominent marginal ridges of the maxillary incisors.
- Sign:** An objective finding or observation made by the examiner that the patient may be unaware of or does not report.
- Sinus:** An airspace inside the skull, such as the maxillary sinus; an abnormal channel, fistula, or tract allowing the escape of pus.
- Smooth surface caries:** Decay affecting the smooth (interproximal, buccal, or lingual) surface of teeth.
- Splenic:** Of or pertaining to the spleen, which is a structure in the upper left abdomen just behind and under the stomach. The spleen contains the largest collection of reticuloendothelial cells in the whole body; its functions include blood formation, blood storage, and blood filtration.
- Spontaneous:** Occurring unaided or without apparent cause; voluntary.
- Squamous epithelium:** An uninterrupted layer of epithelium characterized by the top layer consisting of flat, scalelike cells. Squamous epithelium lines the oral mucosa and skin.
- Stepladder trabecular pattern:** A pattern of medullary bone that appears radiographically as successive linear increments or steps. This pattern is suggestive of extramedullary hematopoiesis that occurs with certain types of anemias.
- Subcutaneous:** Below the cutaneous (epidermal) layer.
- Submucosal palatal cleft:** An incomplete cleft of the palate where the epithelium covers a defect in the subjacent connective tissue.
- Sulcus terminalis:** A shallow V-shaped groove in the dorsum of the tongue behind the circumvallate papillae.
- Superficial:** Located on or near the surface.
- Supernumerary:** In excess of the regular number.
- Supernumerary roots:** An excess number of roots.
- Suppurative:** The discharge of pus from infected tissue.
- Supraeruption:** A condition in which a tooth erupts beyond the normal plane of occlusion. This condition can result when the opposing tooth is missing.
- Symptom:** A manifestation of disease that the patient is usually aware of and frequently reports.
- Syndrome:** A combination of signs and symptoms occurring commonly enough to constitute a distinct clinical entity.
- Talon cusp:** An extra cusp on an anterior tooth that resembles eagle's talon.
- Taurodont:** A malformed, multirrooted tooth characterized by an altered crown-to-root ratio, the crown being of normal length, the roots being abnormally short, and the pulp chamber being abnormally large.
- Taurodontism:** Pertaining to a condition in which there are taurodont teeth.
- Telangiectasia:** The formation of capillaries near the surface of a tissue. Telangiectasia may be a sign of hereditary disorder, alcohol abuse, or malignancy in the region.
- Template bleeding time:** The amount of time necessary for bleeding to stop after a skin incision of consistent length and depth.

- Texture:** Pertains to the characteristics of the surface of an area or lesion. Some descriptions of texture are as follows: smooth, rough, lumpy, and vegetative. The tiny bumps on the surface of a wart cause it to have a vegetative texture.
- Therapeutic:** Of or pertaining to therapy or treatment; beneficial. Therapy has as its goal the elimination or control of a disease or other abnormal state.
- Thorax:** That part of the body between the neck and abdomen, enclosed by the spine, ribs, and sternum. In the vernacular, the thorax is referred to as the chest. The main contents of the thorax are the heart and lungs.
- Thrombocytopathia:** A condition in which there is an abnormality in platelet function.
- Thrombocytopenia:** A condition in which there are less than the normal number of platelets.
- Thrombophlebitis:** The development of venous thrombi in the presence of inflammatory changes in the vessel wall.
- Thrombosis:** Formation of thrombi within the lumen of the heart or a blood vessel. A lumen is the space within a passage; a thrombus is a solid mass that can form within the heart or blood vessels from constituents in the circulating blood. Patients prone to the formation of thrombi should receive anticoagulant therapy.
- Tonsillar pillars (faucet):** The soft tissue folds derived from the second and third branchial arches that border and house the tonsils (lymphoid patches of tissue in the oropharynx).
- Tooth bud:** The embryonic tissue of origin of the teeth; tooth buds develop from the more primitive tissue of the dental lamina.
- Torus:** A bony nodule on the hard palate or on the lingual aspect of the premolars.
- Tourniquet test:** When pressure is applied to the blood vessels of the upper arm using a blood pressure cuff, a bleeding tendency is detected when petechiae develop in the region.
- Trabeculae:** Thin, anastomosing threads of bone or tissue.
- Transient:** Temporary; of short duration.
- Translocation:** Rearrangement of parts. In dentistry, the term is used when a tooth erupts into an abnormal location but remains within the dental arch.
- Translucent:** Somewhat penetrable by rays of light.
- Transposition:** Two teeth exchanging places.
- Trauma:** A wound or injury; damage produced by an external force.
- Treacher Collins syndrome:** An autosomal dominant disorder that results in characteristic head and face abnormalities, which include ear malformations, hearing loss, small jaws, malocclusion, and open bite.
- Trismus:** Tonic contraction of the muscles of mastication; commonly referred to as *lockjaw*. Trismus is caused by oral infections, salivary gland infections, tetanus, trauma, and encephalitis.
- Trunk:** The main part of the body, to which the limbs are attached. The trunk consists of the thorax and abdomen and contains all of the internal organs. This term is also used to describe the main part of a nerve or blood vessel.
- Tumor:** A solid, raised mass that is >1 cm in diameter and has the dimension of depth. This term also describes a mass consisting of neoplastic cells.
- Twinning:** The complete division of a single tooth bud resulting in a divided tooth.
- Ulcer:** Loss of surface tissue caused by a sloughing of necrotic inflammatory tissue; the defect extends into the underlying lamina propria.
- Umbilicated:** Having a central mark or depression.
- Unilateral:** Affecting only one side of the body.
- Uremia:** A toxic condition caused by the accumulation of nitrogenous substances in the blood that are normally eliminated in the urine.
- Urticaria:** A vascular reaction of the skin characterized by the appearance of slightly elevated patches that are either more red or paler than the surrounding skin. Urticaria is also known as hives and may be caused by allergy, excitement, or exercise. These patches are sometimes intensely itchy.
- van der Woude syndrome:** An autosomal dominant syndrome characterized by a cleft lip or cleft palate and distinctive pits of the lower lips or both lips.
- Vasoconstriction:** To decrease the diameter or caliber of a blood vessel.
- Ventral:** Directed toward or situated on the belly surface (opposite of *dorsal*).
- Vermilion:** That part of the lip that has a naturally pinkish red color and is exposed to the extraoral environment. The vermilion contains neither sweat glands nor accessory salivary glands.
- Vermilion border:** The mucocutaneous margin of the lip.
- Vermilionectomy:** Surgical removal of the vermilion border of the lip.
- Vertigo:** An unpleasant sensation characterized mainly by a feeling of dizziness or that one's surroundings are spinning or moving.
- Vesicle:** A well-defined lesion of the skin and mucous membranes that resembles a sac, contains fluid, and is <1 cm in diameter.
- Visceral:** Pertaining to body organs.
- Viscous:** Thick or sticky.
- Wheal:** A localized area of edema on the skin. The area is usually raised and smooth surfaced, and is often very itchy.
- Xerostomia:** Dry mouth.
- X-linked inheritance:** A mode of transmitting a gene carried on the X chromosome from parents to their offspring.

Index

Page numbers followed by *f* denote figures.

- Abfraction, 56, 57*f*
- Abnormalities, by location, 103–128
- Abrasion, 56, 57*f*
- Abscess
 - periapical, 68, 69*f*, 118, 119*f*
 - periodontal, 86, 87*f*, 94, 95*f*, 118, 119*f*
- Accessory salivary gland tumor, 112, 113*f*
- Acquired hypodontia, 44, 45*f*
- Acquired immunodeficiency syndrome. *See* AIDS.
- Actinic cheilosis, 110, 111*f*
- Actinomycotic gingivitis, 84, 85*f*
- Acute apical periodontitis, 90, 91*f*
- Acute atrophic candidiasis, 146, 147*f*
- Addison disease, 152, 153*f*
- Adenomatoid odontogenic tumor, 74, 75*f*
- Agranulocytosis, 100, 101*f*
- AIDS (acquired immunodeficiency syndrome),
 - 182, 183*f*, 184, 185*f*
- Allergic reactions, 166, 167*f*
- Allergic stomatitis, 166, 167*f*
- Alveolar mucosa, frenal attachments and, 6, 7*f*
- Ameloblastic fibro-odontoma, 74, 75*f*
- Ameloblastoma, 74, 75*f*
- Amelogenesis imperfecta, 48, 49*f*
- Anaphylaxis, 166, 167*f*
- Anatomic landmarks, 1–16
 - of oral cavity, 2, 3*f*
 - of periodontium, 6, 7*f*
 - of tongue and variants of normal, 4, 5*f*
- Anatomy
 - of TMJ, 14, 15*f*
 - of tongue, 4, 5*f*
- Anemia, 106, 107*f*
- Anesthetic agents, oral, 198
- Angioedema, 114, 115*f*, 126, 127*f*, 166, 167*f*
- Angular cheilitis, 110, 111*f*, 146, 147*f*
- Ankyloglossia, 4, 5*f*
- Ankylosis, 44, 45*f*, 90, 91*f*
- Anterior lateral region, 10, 11*f*
- Anterior midline region, 10, 11*f*
- Anterior open bite, 14, 15*f*
- Antianxiety agents, 199
- Antibiotic prophylaxis, 192
- Antibiotic therapy, 193
- Antifungal therapy, 195
- Antimicrobial topical agents and rinses, 194
- Antiviral therapy, 196–197
- Atrophy, 26, 27*f*
- Attrition, 56, 57*f*
- Axial tilting, 54, 55*f*
- Bacterial infections, oral, 182, 183*f*
- Behçet syndrome, 174, 175*f*
- Bell palsy, 126, 127*f*
- Benign accessory salivary giant neoplasm, 120, 121*f*
- Bifid uvula, 32, 33*f*
- Bisphosphonate osteonecrosis, 186, 187*f*
- Blandin-Nuhn cyst, 108, 109*f*
- Bleeding, 100, 101*f*, 126, 127*f*
- Body piercing, 108, 109*f*
- Bone loss, 88, 89*f*
- Bony lesions, of jaws, 76, 77*f*
- Botryoid lateral periodontal cyst, 72, 73*f*
- Buccal aspect molar region, 12, 13*f*
- Buccal mucosa, 2, 3*f*
- Buccal space infection, 122, 123*f*
- Bulbous root, 42, 43*f*
- Bulla, 24, 25*f*
- Calcifying epithelial odontogenic tumor, 74, 75*f*
- Calculi, salivary, 116, 117*f*
- Calculus, 82, 83*f*
- Candidal cheilitis, 110, 111*f*
- Candidiasis, 146, 147*f*, 182, 183*f*
- Canine region, 10, 11*f*
- Carcinoma, 28, 29*f*
 - gingival, 94, 95*f*
 - squamous cell, 140, 141*f*, 176, 177*f*
 - verrucous, 134, 135*f*
- Carcinoma syndrome, nevoid basal cell, 72, 73*f*
- Caries. *See* Dental caries.
- Caries progression, 68, 69*f*
- Case studies
 - [1, 2] 16, 16*f*
 - [3, 4] 30, 30*f*
 - [5, 6] 36, 36*f*
 - [7, 8] 62, 62*f*
 - [9, 10] 70, 70*f*
 - [11, 12] 80, 80*f*
 - [13, 14] 102, 102*f*
 - [15, 16] 128, 128*f*
 - [17, 18] 154, 154*f*
 - [19, 20] 178, 178*f*
 - [21, 22] 188, 188*f*
- Cellulitis, 114, 115*f*
- Cementoblastoma, 78, 79*f*
- Cemento-osseous dysplasias, 78, 79*f*
- Central giant cell granuloma, 74, 75*f*
- Cervical resorption, 60, 61*f*
- Cheilitis glandularis, 114, 115*f*
- Cheilosis, actinic and exfoliative, 110, 111*f*
- Chemotherapeutic ulcer, 176, 177*f*
- Children. *See* Oral conditions, affecting infants and children.

- Chronic atrophic candidiasis, 146, 147f
- Chronic hyperplastic candidiasis, 146, 147f
- Cigarette keratosis, 134, 135f
- Cleft lip, 32, 33f
- Cleft palate, 32, 33f
- Cleidocranial dysplasia, 46, 47f
- Clinical applications and resources
- antianxiety agents, 199
 - antibiotic prophylaxis, 192
 - antibiotic therapy, 193
 - antifungal therapy, 195
 - antimicrobial topical agents and rinses, 194
 - antiviral therapy, 196–197
 - common oral lesions, diagnosis and management guide to, 207–226
 - fluoride therapy, 200
 - mucosal ulcer medications, 201–202
 - nutrient deficiency therapy, 203
 - prescriptions and protocols, 191
 - Rx abbreviations, 190
 - saliva substitute, 204
 - sedative/hypnotics, 205
 - tobacco cessation, 206
 - topical oral anesthetic agents, 198
- Commissural lip pits, 32, 33f
- Common oral lesions, diagnosis and management guide to
- papules and nodules, 217–220
 - pigmented lesions, 214–216
 - red lesions, 209–211
 - red-white and, 212–214
 - ulcerative lesions, 224
 - vesiculobullous diseases, 220–223
 - white lesions, 207–209, 225–226
- Compound odontoma, 78, 79f
- Concrescence, 40, 41f
- Condensing osteitis, 76, 77f
- Condyloma acuminatum, 160, 161f, 184, 185f
- Congenital epulis, 34, 35f
- Congenital lymphangioma, 34, 35f
- Contact stomatitis, 166, 167f
- Coronal internal resorption, 60, 61f
- Crossbite, 14, 15f
- Cushing disease and syndrome, 124, 125f
- Cusp of Carabelli, 38, 39f
- Cyclic neutropenia, 100, 101f
- Cystic hygroma, 124, 125f
- Cysts, 24, 25f, 34, 35f, 112, 113f, 116, 117f
 - of Blandin-Nuhn, 108, 109f
 - of jaws, 72, 73f
 - nasopalatine duct, 72, 73f, 118, 119f
 - oral lymphoepithelial, 156, 157f
- Cytomegalovirus, 184, 185f
- Dehiscence, fenestration and, 82, 83f
- Delayed hypersensitivity, 166, 167f
- Dens evaginatus, 38, 39f
- Dens invaginatus, 38, 39f
- Dental anomalies, 37–62
 - root structure, alteration in, 58, 59f, 60, 61f
 - teeth, acquired defects of: noncarious loss of tooth structure, 56, 57f
 - tooth color, alterations in, 52, 53f
 - tooth eruption and tooth position, alterations in, 54, 55f
 - tooth morphology, alterations in, 38, 39f, 40, 41f, 42, 43f
 - tooth numbers, alterations in
 - hyperdontia, 46, 47f
 - hypodontia, 44, 45f
 - tooth structure and color, alterations in, 48, 49f, 50, 51f
- Dental caries, 64, 65f, 66, 67f, 68, 69f
- Dental lamina cysts, 34, 35f
- Dentigerous cyst, 72, 73f
- Dentin dysplasia, 50, 51f
- Dentinogenesis imperfecta, 50, 51f
- Dermoid cyst, 116, 117f
- Desmoplastic fibroma, 92, 93f
- Diabetic gingivitis, 98, 99f
- Diagnostic and descriptive terminology, 18, 19f, 20, 21f, 22, 23f, 24, 25f, 26, 27f, 28, 29f
- Dilaceration, 42, 43f
- Distal drift, 54, 55f
- Drug eruption, lichenoid and lupuslike, 144, 145f
- Drug therapies, systemic, oral manifestations of, 179–188
- Drug-induced gingival overgrowth, 96, 97f
- Drug-induced hyperpigmentation, 186, 187f
- Drugs and therapies, oral effects of, 186, 187f
- Dysplasias, 28, 29f
 - cemento-osseous, 78, 79f
 - cleidocranial, 46, 47f
 - dentin, 50, 51f
 - ectodermal, 44, 45f
 - fibrous, 90, 91f
- Ectodermal dysplasia, 44, 45f
- Ectopic enamel, 42, 43f
- Ectopic eruption, 54, 55f
- Edema, gingival, of hyperthyroidism, 98, 99f
- Emphysema, 126, 127f
- Enamel hypoplasia, 48, 49f
- Endocrine-associated gingival enlargements, 98, 99f
- Ephelis, 148, 149f
- Epstein-Barr virus, hairy leukoplakia and, 184, 185f
- Epulis fissuratum, 94, 95f
- Erosion, 18, 19f, 56, 57f
- Eruption cyst, 34, 35f
- Eruption, of tooth, 54, 55f
- Eruption-related external root resorption, 58, 59f
- Erythema multiforme, 168, 169f
- Erythematous candidiasis, 146, 147f
- Erythroleukoplakia, speckled erythroplakia and, 140, 141f
- Erythroplakia, 140, 141f
- Ewing sarcoma, 124, 125f
- Exfoliative cheilosis, 110, 111f
- Exostosis, 76, 77f
- External coronal resorption, 60, 61f
- External resorption, 58, 59f
- External root resorption, in reimplanted and transplanted teeth, 60, 61f
- Extrinsic staining, 52, 53f
- Face
 - conditions peculiar to, 126, 127f
 - swellings of, 122, 123f, 124, 125f
- False resorption, 58, 59f
- Fenestration, dehiscence and, 82, 83f
- Fibrolipoma, 158, 159f
- Fibromas, 78, 79f, 92, 93f, 158, 159f
- Fibromatosis gingivae, 96, 97f
- Fibro-odontoma, ameloblastic, 74, 75f
- Fibrous dysplasia, 90, 91f
- Fissure, 20, 21f
- Fissured tongue, 4, 5f, 104, 105f
- Florid cemento-osseous dysplasias, 78, 79f
- Fluoride therapy, 200
- Fluorosis, 48, 49f, 52, 53f
- Focal cemento-osseous dysplasias, 78, 79f
- Focal epithelial hyperplasia, 160, 161f

- Focal eruption gingivitis, 84, 85f
 Fordyce granules, 130, 131f
 Frenal attachments, alveolar mucosa and, 6, 7f
 Fungal infections, oral, 182, 183f
 Fusion, 40, 41f
- Gardner syndrome, 46, 47f
 Gemination, 40, 41f
 Generalized anaphylaxis, 166, 167f
 Generalized gingival enlargements, 96, 97f
 Geographic stomatitis, 106, 107f
 Geographic tongue, 106, 107f
 Giant cell fibroma, 158, 159f
 Gingiva
 attached and free marginal, 6, 7f
 periodontium and, disorders of, 81–102
 Gingival bleeding, spontaneous, 100, 101f
 Gingival carcinoma, 94, 95f
 Gingival edema, of hyperthyroidism, 98, 99f
 Gingival enlargements
 endocrine-associated, 98, 99f
 generalized, 96, 97f
 Gingival erythema, linear, 182, 183f
 Gingival lesions, localized, 92, 93f, 94, 95f
 Gingival recession, 82, 83f
 Gingivitis, 84, 85f, 98, 99f, 182, 183f
 leukemic, 100, 101f
 plasma cell, 166, 167f
 Gingivostomatitis, primary herpetic, 96, 97f
 Graft-versus-host disease, 186, 187f
 Granular cell tumor, 108, 109f
 Granuloma
 giant cell
 central, 74, 75f
 peripheral, 92, 93f
 pyogenic, 92, 93f
 Granulomatosis, orofacial, 114, 115f
 Granulomatous ulcer, 176, 177f
- Hairy leukoplakia, 104, 105f
 Epstein-Barr virus and, 184, 185f
 Hairy tongue, 104, 105f
 Hand-foot-and-mouth disease, 164, 165f
 Hard palate, 2, 3f
 Heavy metal pigmentation, 152, 153f
 Hemangioma, 136, 137f
 Hereditary hemorrhagic telangiectasia, 138, 139f
 Herpangina, 162, 163f
 Herpes simplex infection, recurrent, 162, 163f
 Herpes zoster, 164, 165f
 Herpetiform ulceration, 174, 175f
 HIV (human immunodeficiency virus) infection and AIDS, 182, 183f, 184, 185f
 Hormonal gingivitis, 98, 99f
 Human immunodeficiency virus infections and AIDS. *See* HIV infection and AIDS.
 Human papillomavirus, 184, 185f
 Hypercementosis, 42, 43f
 Hyperparathyroidism
 multiple cervical resorption of, 60, 61f
 Paget disease and, 90, 91f
 Hyperpigmentation, drug-induced, 186, 187f
 Hyperplasia, 28, 29f
 focal epithelial, 160, 161f
 lymphoid, 118, 119f
 Hyperthyroidism, gingival edema of, 98, 99f
 Hypertrophy, 26, 27f
 Masseter, 124, 125f
 Hypnotics/sedatives, 205
 Hypocalcified amelogenesis imperfecta, 48, 49f
 Hypodontia, 44, 45f
 Hypomaturation, amelogenesis imperfecta, 48, 49f
 Hypomaturation/hypoplastic, amelogenesis imperfecta, 48, 49f
 Hypoplasia, 26, 27f, 48, 49f
 Hypoplastic amelogenesis imperfecta, 48, 49f
 Hypotrophy, atrophy and, 26, 27f
- Idiopathic osteosclerosis, 76, 77f
 Implantation cyst, 112, 113f
 Incisor-canine region, 12, 13f
 Infants. *See* Oral conditions, affecting infants and children.
 Infections, 122, 123f
 HIV and AIDS, 182, 183f, 184, 185f
 recurrent herpes simplex, 162, 163f
 Infectious mononucleosis, 180, 181f
 Inflammatory external resorption, 58, 59f
 Inflammatory internal root resorption, 60, 61f
 Infrabony defects, 88, 89f
 Internal aspect molar region, 12, 13f
 Intraoral findings
 by color changes, 129–154
 by surface change, 155–188
 Intraorbital space infection, 122, 123f
 Intrinsic discoloration, 52, 53f
 Irritation fibroma, 92, 93f, 158, 159f
- Jaws. *See also* Radiolucent lesions of jaws.
 cysts of, 72, 73f
 radiolucent-radiopaque lesions of, 78, 79f
- Labial, buccal mucosa and, 2, 3f
 Lateral periodontal cyst, 72, 73f
 Lesions. *See also* Pigmented lesions; Red and red-white lesions; Red lesions; Ulcerative lesions; Vesiculobullous lesions; White lesions.
 of jaws, 76, 77f, 78, 79f
 localized gingival, 92, 93f, 94, 95f
 salivary gland, 120, 121f
 Leukemic gingivitis, 100, 101f
 Leukoedema, 131, 131f
 Leukoplakia, 132, 133f
 hairy, 104, 105f
 Lichen planus, 142, 143f
 Lichenoid and lupuslike drug eruption, 144, 145f
 Lichenoid mucositis, 142, 143f
 Linea alba, 130, 131f
 Linear gingival erythema, 182, 183f
 Lingual aspect molar region, 12, 13f
 Lingual thyroid, 108, 109f
 Lingual varicosity, 4, 5f
 Lipoma, 118, 119f, 158, 159f
 Lips, 2, 3f, 114, 115f. *See also* Oral conditions, affecting infants and children.
 conditions peculiar to, 110, 111f
 nodules of, 112, 113f
 Localized anaphylaxis, 166, 167f
 Localized gingival lesions, 92, 93f, 94, 95f
 Ludwig angina, 122, 123f
 Lupus erythematosus, 144, 145f
 Lymphangioma, 160, 161f
 congenital, 34, 35f
 Lymphoepithelial cyst, oral, 156, 157f
 Lymphoid hyperplasia, 118, 119f
 Lymphoma, 118, 119f
 Non-Hodgkin, 184, 185f

- Macrodonia, 38, 39f
- Macroglossia, 104, 105f
- Macule, 18, 19f
 - oral melanotic, 150, 151f
- Major aphthous, 174, 175f
- Malignancies, oral, 184, 185f
- Malignant accessory salivary giant neoplasm, 120, 121f
- Malignant disease-related periodontal defect, 90, 91f
- Malocclusion. *See* Occlusion.
- Mandible, regions of, 12, 13f
- Mandibular tori, 76, 77f
- Masseter hypertrophy, 124, 125f
- Masseteric space infection, 122, 123f
- Maxilla, regions of, 10, 11f
- Medications, mucosal ulcer, 201–202
- Melanoma, 150, 151f
- Melanoplakia, 148, 149f
- Melanotic neuroectodermal tumor, of infancy, 34, 35f
- Mesenchymal nodules and tumors, 112, 113f
- Metaplasia, 28, 29f
- Metaplastic internal root resorption, replacement relating to, 60, 61f
- Meth mouth, 186, 187f
- Microdonia, 38, 39f
- Migration, 54, 55f
- Molar regions, 10, 11f, 12, 13f
- Mononucleosis, infectious, 180, 181f
- Morsicatio buccarum, 130, 131f
- Mottled enamel, fluorosis of, 48, 49f
- Mouth breathing, gingivitis relating to, 84, 85f
- Mouth, floor of, 2, 3f
 - swellings of, 116, 117f
- Mucocele, 112, 113f, 116, 117f
- Mucolingual junction, 6, 7f
- Mucosal ulcer medications, 201–202
- Multiple cervical resorption, of hyperparathyroidism, 60, 61f
- Myxoma, 74, 75f

- Nasolabial cyst, 112, 113f
- Nasopalatine duct cyst, 72, 73f, 118, 119f
- Natal teeth, 34, 35f
- Necrotizing periodontal disease, 182, 183f
- Necrotizing sialometaplasia, 121, 121f
- Necrotizing ulcerative gingivitis, 84, 85f, 182, 183f
- Necrotizing ulcerative periodontitis, 182, 183f
- Neoplasm, accessory salivary giant, benign and malignant, 120, 121f
- Neurofibroma, 158, 159f
- Neurofibromatosis, 124, 125f
- Neutropenia, cyclic, 100, 101f
- Nevoid basal cell carcinoma syndrome, 72, 73f
- Nevus, 150, 151f
 - White sponge, 132, 133f
- Nicotine stomatitis, 134, 135f
- Nodules, 22, 23f, 156, 157f, 158, 159f
 - of lips, 112, 113f
 - papules and, 217–220
- Non-Hodgkin lymphoma, 184, 185f
- Nonvital teeth, 52, 53f
- Normal anatomy, of TMJ, 14, 15f
- Normal, definition of, 26, 27f
- Normal opening, of TMJ, 14, 15f
- Nutrient deficiency therapy, 203

- Occlusion
 - malocclusion and, classes of, 8, 8f, 9f
 - traumatic, orthodontic tooth movement relating to, 90, 91f
- Odontoameloblastoma, 74, 75f
- Odontodysplasia, regional, 50, 51f
- Odontogenic fibroma, peripheral, 92, 93f, 158, 159f
- Odontogenic infection, 122, 123f
- Odontogenic keratocyst, 72, 73f
- Odontogenic tumor, 74, 75f
- Odontoma, 78, 79f
- One-wall infrabony defect, 88, 89f
- Open contact and poor restoration contour, 88, 89f
- Opening, of TMJ, deviation on, 14, 15f
- Oral anesthetic agents, topical, 198
- Oral bacterial infections, 182, 183f
- Oral cavity, 2, 3f
- Oral conditions, affecting infants and children, 32, 33f, 34, 35f
- Oral erythema multiforme, 168, 169f
- Oral fungal infections, 182, 183f
- Oral lesions. *See* Common oral lesions, diagnosis and management guide to.
- Oral lymphoepithelial cyst, 156, 157f
- Oral malignancies, 184, 185f
- Oral manifestations, of sexual conditions and systemic drug therapies, 179–188
- Oral melanotic macule, 150, 151f
- Oral squamous papilloma, 160, 161f
- Oral viral infections, 184, 185f
- Orofacial granulomatosis, 114, 115f
- Oropharynx, tonsils and, 2, 3f
- Orthodontic external root resorption, 60, 61f
- Orthodontic tooth movement, 54, 55f
 - traumatic occlusion and, 90, 91f
- Ossifying fibroma, 78, 79f
 - peripheral, 92, 93f
- Osteitis, condensing, 76, 77f
- Osteoma, 76, 77f
- Osteosclerosis, idiopathic, 76, 77f
- Overhang, 88, 89f

- Paget disease and hyperparathyroidism, 90, 91f
- Palatal tori, 76, 77f
- Palatal torus, 118, 119f
- Palate
 - cleft, 32, 33f
 - hard, 2, 3f
 - soft, 2, 3f
 - swellings of, 118, 119f
 - salivary gland lesions, 120, 121f
- Palato-gingival groove, 40, 41f
- Papules, 22, 23f
 - nodules and, 217–220
- Papulonodules, 160, 161f
- Paradental cyst, 72, 73f
- Paramedian lip pits, 32, 33f
- Parotid, 2, 3f
- Partial eruption, 54, 55f
- Parulis, 34, 35f, 94, 95f
- Patch, 18, 19f
- Pemphigoid, 170, 171f
- Pemphigus vulgaris, 170, 171f
- Periapical abscess, 68, 69f, 118, 119f
- Periapical cemento-osseous dysplasias, 78, 79f
- Periapical inflammation, 68, 69f
- Periapical radiopacities, 76, 77f
- Pericoronitis, 94, 95f
- Periodontal abscess, 86, 87f, 94, 95f, 118, 119f
- Periodontal cyst, lateral, 72, 73f
- Periodontal defect, malignant disease-related, 90, 91f
- Periodontal disease, 22, 23f, 82, 83f
 - necrotizing, 182, 183f
 - radiographic features of, 88, 89f
- Periodontal ligament space, lamina dura and, 90, 91f

- Periodontitis, 86, 87f
 acute apical, 90, 91f
 necrotizing ulcerative, 182, 183f
- Periodontium, 6, 7f
 gingiva and, disorders of, 81–102
- Peripheral giant cell granuloma, 92, 93f
- Peripheral odontogenic fibroma, 92, 93f, 158, 159f
- Peripheral ossifying fibroma, 92, 93f
- Peutz-Jeghers syndrome, 152, 153f
- Pigmentation, heavy metal, 152, 153f
- Pigmented lesions, 148, 149f, 150, 151f, 152, 153f, 214–216
- Plaque, 22, 23f, 82, 83f
- Plasma cell gingivitis, 166, 167f
- Polyp, pulp, 68, 69f
- Posterior open bite-midline deviation-crossbite, 14, 15f
- Postoperative bleeding, 126, 127f
- Premolar region, 10, 11f
- Prescriptions, protocols and, 191
- Primary herpetic gingivostomatitis, 96, 97f
- Prophy paste gingivitis, 84, 85f
- Prophylaxis, antibiotic, 192
- Protocols, prescriptions and, 191
- Protostylid, 38, 39f
- Pseudoaphthous, 172, 173f
- Pseudomembranous candidiasis, 146, 147f, 182, 183f
- Pulp polyp, 68, 69f
- Purpura, 100, 101f, 136, 137f
- Pustule, 24, 25f
- Pyogenic granuloma, 92, 93f
- Radiographic alterations, of periodontal ligament and lamina dura, 90, 91f
- Radiographic features, of periodontal disease, 88, 89f
- Radiographic landmarks
 of mandible, 12, 13f
 of maxilla, 10, 11f
- Radiolucent lesions of jaws
 bony, 76, 77f
 cysts, 72, 73f
 periapical radiopacities, 76, 77f
 tumors, 74, 75f
- Radiolucent-radiopaque lesions of jaws, 78, 79f
- Ranula, 116, 117f
- Recession, gingival, 82, 83f
- Recurrent aphthous stomatitis, 172, 173f
- Recurrent dental caries, 66, 67f
- Recurrent herpes simplex infection, 162, 163f
- Red and red-white lesions, 140, 141f, 142, 143f, 144, 145f, 146, 147f, 212–214
- Red lesions, 136, 137f, 138, 139f, 209–211
- Regional odontodysplasia, 50, 51f
- Reimplanted and transplanted teeth, external root resorption in, 60, 61f
- Replacement/metaplastic internal root resorption, 60, 61f
- Resorption, 58, 59f, 60, 61f
- Retrocuspid papilla, 156, 157f
- Root resorption, 58, 59f, 60, 61f
- Root structure, alteration in, 58, 59f, 60, 61f
- Roots
 bulbous, 42, 43f
 dental caries relating to, 66, 67f
 supernumerary, 42, 43f
- Rotated teeth, 54, 55f
- Rx abbreviations, 190
- Saliva substitute, 204
- Salivary calculi, 116, 117f
- Salivary duct cyst, 116, 117f
- Salivary gland lesions, 120, 121f
- Scalloped tongue, 104, 105f
- Scar, 20, 21f
- Scleroderma, 90, 91f
- Sedative/hypnotics, 205
- Sequelae, dental caries and, 68, 69f
- Sexual conditions, systemic drug therapies and, oral manifestations of, 179–188
- Sexually related and sexually transmissible conditions, 180, 181f
- Sexually transmitted pharyngitis, 180, 181f
- Shovel-shaped incisor syndrome, 42, 43f
- Sialadenitis, 120, 121f
- Sialadenosis, 124, 125f
- Simple bone cyst, 72, 73f
- Sinus, 20, 21f
- Sjögren syndrome, 124, 125f
- Smoker's melanosis, 148, 149f
- Snuff dipper's patch, 134, 135f
- Socket sclerosis, 76, 77f
- Soft palate, 2, 3f
- Speckled erythroplakia, erythroleukoplakia and, 140, 141f
- Spontaneous gingival bleeding, 100, 101f
- Squamous cell carcinoma, 140, 141f, 176, 177f
- Squamous papilloma, oral, 160, 161f
- Stevens-Johnson syndrome, 168, 169f
- Stomatitis, 166, 167f
 geographic, 106, 107f
 nicotine, 134, 135f
 recurrent aphthous, 172, 173f
- Sturge-Weber angiomatosis, 138, 139f
- Supernumerary roots, 42, 43f
- Supraeruption, 54, 55f
- Swellings
 of face, 122, 123f, 124, 125f
 of mouth floor, 116, 117f
 of palate, 118, 119f, 120, 121f
- Syphilis, 180, 181f
- Systemic drug therapies, oral manifestations of, 179–188
- Talon cusp, 38, 39f
- Tattoo, 148, 149f
- Taurodontism, 42, 43f
- Teeth
 acquired defects of: noncarious loss of tooth structure, 56, 57f
 natal, 34, 35f
 nonvital, 52, 53f
 reimplanted and transplanted, external root resorption in, 60, 61f
 rotated, 54, 55f
- Tempromandibular joint. *See* TMJ.
- Tetracycline staining, 52, 53f
- Therapy. *See* clinical applications and resources.
- Three-wall infrabony defect, 88, 89f
- Thrombocytopathic, thrombocytopenic purpura and, 100, 101f
- Thrombocytopenic purpura, 100, 101f
- Thrombus, 136, 137f
- Thrush, 34, 35f
- TMJ (tempromandibular joint), 14, 15f
- Tobacco cessation, 206
- Tobacco-associated white lesions, 134, 135f
- Tongue
 conditions peculiar to, 104, 105f, 106, 107f, 108, 109f
 fissured, 4, 5f, 104, 105f
- Tonsils, oropharynx and, 2, 3f
- Tooth color, alterations in, 52, 53f
- Tooth eruption, tooth position and, alterations in, 54, 55f
- Tooth morphology, alterations in, 38, 39f, 40, 41f, 42, 43f
- Tooth movement, orthodontic, 54, 55f

- Tooth numbers, alterations in
 - hyperdontia, 46, 47*f*
 - hypodontia, 44, 45*f*
- Tooth structure
 - and color, alterations in, 48, 49*f*, 50, 51*f*
 - noncarious loss of, 56, 57*f*
- Topical oral anesthetic agents, 198
- Torus, exostosis, and osteoma, 156, 157*f*
- Toxic epidermal necrolysis, 168, 169*f*
- Translocation, 54, 55*f*
- Transplanted teeth, external root resorption in, 60, 61*f*
- Transposition, 54, 55*f*
- Trauma, of lips, 114, 115*f*
- Traumatic conditions, sexually related conditions relating to, 180, 181*f*
- Traumatic neuroma, 158, 159*f*
- Traumatic occlusion, orthodontic tooth movement and, 90, 91*f*
- Traumatic ulcer, 172, 173*f*
- Traumatic white lesions, 132, 133*f*
- Tuberosity region, 10, 11*f*
- Tumors, 22, 23*f*, 112, 113*f*
 - granular cell, 108, 109*f*
 - of jaws, 74, 75*f*
 - melanotic neuroectodermal, of infancy, 34, 35*f*
 - Warthin, 124, 125*f*
- Turner's tooth, 48, 49*f*
- Twinning, 40, 41*f*
- Two-wall infrabony defect, 88, 89*f*
- Ulcer, 18, 19*f*
 - chemotherapeutic, 176, 177*f*
 - granulomatous, 176, 177*f*
 - mucosal, medications for, 201–202
 - traumatic, 172, 173*f*
- Ulceration, herpetiform, 174, 175*f*
- Ulcerative gingivitis, necrotizing, 84, 85*f*, 182, 183*f*
- Ulcerative lesions, 172, 173*f*, 174, 175*f*, 176, 177*f*, 224
- Ulcerative periodontitis, necrotizing, 182, 183*f*
- Unicystic ameloblastoma, 74, 75*f*
- Varicella, 164, 165*f*
- Varicella-zoster virus, 184, 185*f*
- Varicosity, 136, 137*f*
 - lingual, 4, 5*f*
- Verruca vulgaris, 160, 161*f*
- Verrucous carcinoma, 134, 135*f*
- Vesicle, 24, 25*f*
- Vesiculobullous diseases, 220–223
- Vesiculobullous lesions, 162, 163*f*, 164, 165*f*, 166, 167*f*, 168, 169*f*, 170, 171*f*
- Viral infections, oral, 184, 185*f*
- Warthin tumor, 124, 125*f*
- Wheal, 20, 21*f*
- White lesions, 130, 131*f*, 132, 133*f*, 207–209, 225–226
 - tobacco-associated, 134, 135*f*
- White sponge nevus, 132, 133*f*